# CONTRIBUTIONS TO THE STUDY

OF THE

# HEART AND LUNGS.



BY

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NEW YORK:
E. B. TREAT, 771 BROADWAY.
1887. Price, $2.75.

TO THE MEMORY

OF

GEORGE PHILIP CAMMANN, M.D.,

OF NEW YORK,

A PUPIL OF LOUIS, ONE OF THE EARLIEST AUSCULTATORS OF THIS COUNTRY, MY TEACHER, THESE MONOGRAPHS ARE REVERENTLY DEDICATED.

# PREFACE.

THESE monographs, which appeared at different times in Medical Journals and in Transactions of Medical Societies, are here collected with dates and places of first appearance given.

They are divided into three parts.

First: those pertaining to the respiratory organs.

Second: those pertaining to the heart.

Third: those pertaining to miscellaneous subjects, having some relevancy to parts 1st and 2d.

The all-prevailing benevolence of the medical profession prevents it from acknowledging any *patent right in ideas.*

Any great principle or truth discovered, immediately becomes common property.

So much the more it is due that just mention should be made of the originator or discoverer.

In the past this justice has been done. We see it in names applied to diseases, to portions of anatomy and to methods of operations, as well as by statements of writers on special subjects as to priority of authorship.

It may seem unnecessary here to make any claim of novelty as to some of the views or positions maintained, but I am certain there are some who will agree with me that at the time of their first appearance they were met by hostile criticism, in nearly all of their distinctive points. And also that at the present time some of these points have been adopted by writers and teachers without acknowledgment.

It makes but little difference to whom is accredited whatever may be worthy of adoption, yet it is pleasant to know that our labor has received an appreciative approval from those who are worthy to give it.

J. R. LEAMING.

NEW YORK. 18 West 38th Street.
*January*, 1887.

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# PART I.

## LUNGS AND PLEURA.

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### INTRODUCTION.

THE important points in which originality is claimed in this first division are—

*First:* The analysis of respiratory murmurs and sounds.

*Second:* Pointing out the existence and diagnostic value of true respiratory murmurs.

*Third:* The constitution and resistance of residual air.

*Fourth:* The site and mechanism of râles and rhonchi.

*Fifth:* Recognizing the importance and special office of the nutrient artery, and the philosophy of its agency in producing and in removing pathological results.

These points are intimately related to and dependent upon each other.

They form an outline of a body of doctrine in regard to the causation, pathology, physical signs, diagnosis and treatment of disease of the respiratory system.

We cannot make an analysis of any diagnostic value of the respiratory organs without considering the persistent occupancy of the true respiratory system by

residual air. Its chemical constitution and forcible resistance to displacement, by inspired air, not permitting currents to take place. The attraction of affinity between the air molecules and the blood globules keep up a constant molecular movement within the mass of compressed residual air. The pure air molecules are attracted to the blood globules, and the blood globules are attracted to the air molecules, and each tries to get to the other, and this results in a constant circulation. The blood globule moves along within the capillary until it has made its exchange, giving off carbonic acid gas, and detritus, and receiving oxygen in return, and now it is pushed along through the venous radicle until within the pulmonary vein, and the whole mass is moving as arterial blood into the left heart to enter the systemic circulation.

The air molecule attracted by the blood globule rushes among other molecules, pushing them aside and backwards until it reaches the globule within its capillary, when it gives off its oxygen, and receives in return the carbonic gaseous refuse, when, losing its attraction, it is pushed back in turn by the eager molecules still under attraction, until it is high up in the bronchia, to be thrown out into the air by expiration.

The air sacs contract upon the residual air, compressing it, while the molecules under the influence of heat continually and forcibly expand. The muscular susurrus is resonated in the air sacs, producing a low murmur, continuous but increasing in loudness during inspiration, and decreasing during expiration. This murmur measures the capacity of the lungs for aëration of the blood. It is lost in emphysema, and is feeble in phthisis or disappears

entirely. This murmur so characteristic is confined to the true respiratory system: I have named it the true respiratory murmur; and the friction of air moving in a body through the mouth, nose, pharynx, larynx and bronchæ, as broncho-respiratory murmur.

We have a better comprehension of the qualities of residual air after considering the philosophy of respiratory murmurs, and especially of that of the true respiratory. We must remember that the air does not go through the true respiratory system, down into and through the smaller bronchi and bronchioli into the air sacs and back again, as the older writers have taught us.

But sound vibrations do go on through the compressed resonating residual air, forcibly distending the air sacs all the more readily. Let the auscultator place his ear or a stethoscope against the chest wall and listen. If the lung is in good condition and not adherent, it will move quite a distance while the ear is stationary. Each little air sac going to make up a lobulette is a resonator and is in sound connection with the open air.

During the inspiration many of these lobulettes pass under the ear in rapid succession, and give rise to the smooth, even vibrations which denote health. If the lung is adherent, of course it cannot move under the ear, and we say there is feeble respiration; or if some of the air sacs are dilated or some of the bronchioli are obstructed, or if there is viscid secretion or lymph deposits or any other cause of unusual conduction, we say the respiration is harsh or bronchial, and there is a loss of true respiratory murmur. We have learned practically to analyze the respiratory murmurs. Where there is doubt we ask the patient to take a full

inspiration and then hold the breath. If the true respiratory murmur can be heard at all we know that aëration of the blood is taking place, and we measure the degree by its fullness or feebleness.

If there is absence of the true respiratory murmur the aëration of the blood is imperfectly performed, and the patient is unable to hold the breath but momentarily. But by breathing with frequent inspirations, he endeavors to make up for fullness of expansion, and the velocity of the movement of the tidal air raises the pitch, and gives it a harshness which we call puerile respiration, because it is like the respiratory murmur in children before the true respiratory system is developed. The older auscultators following Laennec, especially Andral, called the perfect unison and harmony of the combined broncho-respiratory and the true respiratory, the *vesicular murmur*, supposing that the murmur was formed by air friction within the air sacs or vesicles, not recognizing the impossibility of two bodies occupying the same space at the same time.

They did not recognize residual air nor its varying quantity and quality in health and in disease.

In rapid frequent respirations the amount of residual air is diminished. So it is in fevers, in Bright's disease, in Asiatic cholera, or in any condition in which the quality of blood is so altered as to lose affinitive attraction for the air. In all such cases the tidal air goes farther into the true respiratory system, in the same degree as the residual air is diminished, and then its resistance is more easily overcome. The broncho-respiratory murmur is then in excess.

By clearly understanding the foregoing principles of respiratory murmurs, and of the constitution and resistance of the residual air, we may come to have a

clear idea of the mechanism and site of râles and rhonchi. We see that the residual air prevents currents in the true respiratory system even when its amount is diminished by disease; it still guards the aërating functions. If there are no currents of air there can be no breaking of bubbles, nor separation of agglutinated air sacs or vesicles.

Louis says the size of the râle measures the size of the tube or of the vesicle in which it is formed. And upon that reasoning, the crepitant râle is placed within the vesicles, meaning the air sacs, and not the cup-shaped open-mouthed alveoli found in the walls of the sacs, or in the terminal bronchioli.

But if the size of the râle measures the size of the tube or air sac or vesicle, even the crepitant râle, the smallest which we hear, is many times larger than the largest air sac.

The site of these râles cannot be within the true respiratory system, for the residual air prevents, and the size of the râle is proof that it has a different site and mechanism; it can only be within the pleuræ, for the rales are often heard when the lung is consolidated. All of the smaller râles have an inter-pleural origin, and also most of the larger.

Inter-bronchial or mucous râles have their site in the bronchial or convective system, in the path of the tidal air.

They are nearly always intermittent, and disappear when the mucous is expectorated, or the cause removed.

These mucous râles are always large and have a liquid quality of sound, as have also some of the inter-pleural râles in which small collections of fluid are imprisoned among the adhesions. The intermittent character of mucous or inter-bronchial râles is a

marked characteristic. The râle is heard over a larger space, according to the extent and direction of the bronchial tube. It may be traced to several different points where the sound is conducted into the chest wall. Inter-pleural râles are heard only in the immediate neighborhood of their origin. They are never heard over a large space, and they are not intermittent as are the inter-bronchial. They are near the ear, and even when they have the mucous liquid gurgle, they are not far away, and generally the auscultator can hear the respiratory murmurs beyond the râles. Being accustomed to making the distinction, he detects the difference at once.

Direct conduction of sound vibrations from within into the chest wall, is like opening a window into a dark room, which brings into view things that before were concealed.

The heart is mostly covered with lung, which is a non-conductor of sound. It has indirect connection with the chest wall by its attachments, which keep it in its place. The apex has greater freedom of movement, and in performing its function is brought directly in contact with the chest wall at the point of apex beat only. Hence we hear the heart sounds and murmurs more clearly. The lungs are a mass of convective tubes and air sacs running in slightly diverging lines from their origin in the trachea, and they completely fill up and *pack* the cavity of the chest. The acoustic chamber is capable of great expansion by reason of the diaphragm below, and the cartilage of the ribs in front attached to the sternum, a movable sound-board. Air is the universal medium of sound, and vibrations commenced in this medium pass in waves from the point of origin in every direction until they

are arrested by a non-conductor, such as a distensible bronchi or air sac. Sound vibrations in air in tubes pass in one direction only, in their length. Consequently the lungs are a poor conductor of sound, except in one direction, in that of the myriad columns of air confined in tubes. The air sac, which is the distal end of each minute column of sound vibrative air, is pressed against the chest wall and delivers its vibrations into it. But in the act of respiration the air sac travels over and back again quite a little space of chest wall, delivering its sound-vibrations as it goes. The ear being stationary against the outer surface of the chest wall, like a general reviewing his army, hears column after column salute as it passes, and takes notice of every variation from the normal. The source of sound in the heart is in its contraction. The source of sound in the lungs is in the columns of air, speaking-tubes in connection with the outer world. But attachments of the lungs to the pericardial sac, or to the chest wall, makes direct sound-connection, which becomes audible in the area of connection. But the connection itself is also a source of sound in friction and stretching the adhesions that have taken place. Adhesions in time contract and close up many of the peripheral air sacs, contracting the lung and drawing in the chest wall, creating new and permanent areas of unusual sound conduction. Inspection of the chest wall will reveal these areas to the sight. When the chest expands these areas will apparently be drawn in. When these areas are over condensations of lung tissue extending to the larger bronchæ, the sound simulates that of a cavity in a remarkable degree, and is the source of false diagnosis. These condensations of lung tissue, attached by a strong band to the chest wall, are liable not only to

deceive the auscultator but the pathologist, by being mistaken for cicatrices of closed cavities.

The peculiarity of the minute anatomy of the nutrient artery of the true respiratory system, is of great interest physiologically and pathologically.

The nutrient artery is derived from the systemic circulation and ends in the pulmonic.

It is a *short cut* between. It is an anomaly. In every other artery the blood, after being distributed to the capillaries, is gathered up as venous blood by the venous radicles of the accompanying vein, and is sent to the right heart to be sent thence again to the lungs for aëration. The blood of the nutrient artery is not sent again to the right heart, but is gathered up by the venous radicles of the pulmonary vein as arterial blood, and is sent at once to the left heart to be thrown again into the systemic circulation. Unlike any other artery in the body it has no venæ comites to return the blood to the right heart for pulmonary circulation. The capillary radicles of the pulmonary vein gather up the blood of the nutrient artery as well as that of the pulmonary artery after passing through the capillaries of the true respiratory system, where both are aërated and prepared for systemic circulation, and are together sent to the left heart. The venous radicles of the pulmonary vein gather up the aërated blood both from the pulmonary capillaries and the capillaries of the nutrient artery, and carry all to the left heart. Hence any obstruction of the capillaries of the true respiratory system must throw the blood back upon two sources, that of the pulmonary artery and of the nutrient artery derived from the bronchial. The obvious result is hyperæmia of the lungs and fullness of the nutrient and bronchial arteries. Nature's relief for these conditions is exuda-

tions upon the pleural surface of the lungs, and of mucous or blood within the bronchæ, sometimes taxing the auscultator to diagnosticate the difference. But as the blood or mucous is exuded only upon the mucous membrane above the residual air, it is not difficult to do.

## ARTICLE I.

### Remarks made before the New York Academy of Medicine, in discussing Dr. Alonzo Clark's Paper on Pneumonia.*

Mr. President: In common with many others, I desire to express my indebtedness to Dr. Clark for the knowledge that "the exudative matter in pneumonia is not puriform, and does not become so even in the stage of gray hepatization;" which is of the highest practical importance, and of which I have availed myself many times.

As to the seat of the inflammatory action, and also as to the significance of the physical sign of crepitant râle, I have formed independent opinions, not entirely in accordance with those expressed by Dr. Clark, nor with those held by the profession generally; and I should hesitate to advance them against such high authority, did I not believe that the cause of truth may be benefited by stating the reasons upon which they are founded.

I understand Dr. Clark to maintain that the seat of disease in inflammation of the lungs is confined to the lining membrane of the air-cells; and the proof alleged is that in post-mortem examinations the exuded matter is always found occupying the air-cells alone, the cellular connective tissue being void of pathological change. This statement I do not question, but I do believe that there is a time in the course of the disease when the fibrous connective tissue is the subject of inflammation. The natural cure of inflammation is exudation;

* October, 1865.

but interstitial exudation would endanger the life of this important but delicate portion of the body; consequently we find the exuded matter poured into the air cells, where it is comparatively innocuous—and this is not a singular provision, the same thing is done in inflammation of the pleura, for instance, when the exuded serum is poured into its cavity. Ought we to expect to find interstitial exudation as proof of past inflammation in the connective tissue, when the cure has already been completed by abundant exudation into the free and capacious true respiratory system? It seems to me that finding it here is in itself strong evidence that inflammation has previously existed in the fibrous connective tissue.

The air-passages are lined with ciliated epithelial mucous membrane, and simple inflammation of this membrane is not followed by exudation; but if the inflammation extends to the subjacent tissues, which are fibrous, exudation is the natural result; and, further, the character of the underlying fibrous tissue determines the kind of exudation; as, above the epiglottis it may be diphtherial, while below it will be croupal. The mucous membrane being the same, the difference in the exudation must be owing to the special conditions of the underlying tissues. If, then, the mucous membrane lining the larger air-passages acts merely as a strainer, and has nothing to do with the formation of the exuded pseudo-membrane, why should not the matter exuded through the tessellated pavement epithelium of the true respiratory system be regarded as the result only of inflammation of connective tissues.

Dr. Clark speaks of the sign of crepitant râle as denoting the fact of exudation, and as being caused by it. To this view I dissent. Crepitant râle is a sign of great importance as marking one stage of the disease,

but not that of exudation. The physical signs in pneumonia are inseparably connected with the pathological changes which give them existence, and during an ordinary attack of uncomplicated pneumonia their regular succession is as follows: during the stage of engorgement there is muffling of the true respiratory sound, with slightly exaggerated broncho-respiratory murmur, the percussion note being a trifle raised in pitch.

The second change is denoted by crepitant râle, the disappearance of the true respiratory sound, more exaggeration of the broncho-respiratory murmur, the percussion note still further raised in pitch, but without loss of resonance.

The third change is marked by disappearance of crepitus and the appearance of tubular breathing; the percussion note is dull, flat, raised in pitch, with great loss of resonance. These signs remain till resolution commences, when they gradually disappear, and those of health take their place.

The first change is accompanied by chilly sensations, pains in the head, back and limbs, an accelerated bounding pulse, and oppressed respiration. The second change, where crepitus is present, the breathing is hurried rather than oppressed, the skin is hot and dry, the pulse more frequent, harder, and smaller, and occasionally there is mental disturbance. The third change, where the crepitus disappears and tubular breathing takes its place, with dullness under percussion, the activity of the symptoms notably subsides; the respiration, though still frequent, is not so hurried; the pulse is softer, fuller, and slower; the skin loses its heat, and sometimes becomes moist. The second change, where crepitus is present, I regard as inflammatory, with its seat in the fibrous connective

tissue. The third change indicates the natural cure by exudation. In complicated pneumonia this regular order of signs and symptoms is interfered with, and sometimes reversed. In broncho-pneumonia of the travelling kind, we may have signs of the process of natural cure going on in one small portion of lung, while an adjacent part may be undergoing the first or second change. The respiratory murmur is composed of two elements—the broncho-respiratory, formed by the tidal air in the convective tubes, and the true respiratory, having its origin alone in the true respiratory system. They differ in origin, character, and quality, and may be studied separately as well as in combination.

The lung is composed of a convective system and the true respiratory system. The convective system is composed of the bronchial tubes and air-passages, and is only slightly distensible. The true respiratory system is composed of the terminal bronchi, or the air-sacs, and is characterized by the presence of alveoli, and is immensely distensible. These two systems differ anatomically, structurally, and functionally. In unhurried healthy respiration the air enters in a body into the bronchi as far as the third or fourth division, when it is instantly mixed with the residual air, becoming a component part of it, and by its addition dilating equally the distensible true respiratory system. These physiological facts are pretty well established in the minds of competent observers, and being admitted preclude the idea of currents in the residual air rushing into air-sacs and out again through intralobular passages.

I can conceive of no action or motion in the residual air save the molecular, which is governed, first, by the law of the diffusion of gases, and, second, by that of

affinitive attraction. The newly-introduced atmospheric air, being diffused throughout the residual air, now comes under the influence of the law of affinitive attraction; and each separated molecule struggles toward the lining membrane of an alveolus to meet a blood-globule, which is also struggling along the network of capillaries, under the same propelling influence of affinitive attraction, gives up its oxygen, receives effete matter in exchange, loses its affinity, is repelled, crowded back by other struggling air particles, till it is forced far up in the bronchia, and thence is expired.

The presence of muscular fibre in the delicate tissues of the true respiratory system is not yet established beyond all cavil; but it is certain that the air-sacs have power of resistance and active contraction, qualities belonging to muscle, of which fact we are painfully sensible in its loss in vesicular emphysema.*

If the ear be placed over the lung of a healthy young person during unhurried respiration, and the auscultatory signs be carefully analyzed, the observer will be conscious of two elementary sounds in the respiratory murmur. The broncho-respiratory or tidal-air sound will be heard almost entirely alone in inspiration, and will be recognized as air-friction sound, is of moderately high pitch and slightly harsh in character, and has been likened to the gentle rustling of the leaves of a tree stirred by the breeze. The other, which is the true respiratory element, is a soft, gentle murmur, low in pitch, heard both in inspiration and expiration, swelling in the one and diminishing in the other, is continuous, and may be heard alone when the patient

* It is now established that muscular fibre extends over the air cells.

holds his breath after a full inspiration. It may be compared to the roar of the sea heard at a great distance.

It differs in pitch and quality from all other respiratory sounds, and impresses the mind with the idea of an infinite number of regular vibrations. Its separate identity and distinctive qualities may be studied to best advantage when all other sounds are cut off in the air-passages by holding the breath. It is not a characterless noise, but is a sound subject to definite acoustic law, and its alteration or diminution is the earliest sign we have of approaching pulmonary disease. It is not readily heard: an acute ear, even after considerable auscultatory education, is necessary to its discriminating analysis being of the highest diagnostic value. In the congestive stage of pneumonia it is muffled and obscured, and when crepitus gives evidence of the second change it disappears.

Whatever may be considered as the cause of the true respiratory sound, it is only coexistent with a healthy condition of the true respiratory system. At the first invasion of inflammation the true respiratory system loses its quality of distensibility, and each inspiration afterward, suddenly increasing the volume of residual air, forcibly distends the altered and stiffened air-sacs and alveoli, causing the fine crackling sound of crepitant râle.* This is my understanding of the seat of inflammation in pneumonia, and the significance of the sign of crepitant râle.

The practical teaching of these views, connected with the knowledge, for which we are so much indebted to Dr. Clark, of the discovery of the non-

* In later papers the seat of crepitant râle is placed in the pleura.

puriform character of the exuded matter in pneumonia, is, that treatment may certainly abort the disease in the first, or stage of engorgement, and that it is frequently possible in the second, or stage of active inflammation, denoted by crepitus; at all events, before consolidation, the disease may be modified and shortened, adding much to the safety of the patient. But when the third or exudative stage has taken place, the duty of the physician will be confined mostly to hygiene and intelligent observation.*

* More extended experience has proved to me that the seat of crepitant râle is almost always interpleural.

The above explanation may be competent in a degree after the lung has become adherent to the chest wall; but centric pneumonia without interpleural plastic exudation is unaccompanied with râles, either crepitant or sub-crepitant, nor is there bronchial breathing. And, again, crepitant râle may exist without pneumonia, as when interplural adhesions are very close and do not allow of but slight movement of the lung almost at the end of forced respiration, when there will be a little shower of crepitant râle.

## ARTICLE II.

### Pleuritis.*

Mr. President: I desire to restate some of the points in the paper under discussion—to make some explanations, to give some instances, in order that its meaning may not be misunderstood.

The underlying thought in the paper, which caused it to be written, is, that pleuritic adhesions confine the motion of the lung, cause systemic irritation, accelerate the heart-beat, disturb the digestive function, lower the vital power, and render the occurrence of active phthisis probable, especially where there is inherited tubercular predisposition. My experience in public institutions and private practice, which is somewhat extended, has fixed the thought in my mind, and ever since it has taken shape I have been at pains to verify the opinion in physical examinations and at autopsies. I have not kept full statistical tables, yet I can truly state that in a large proportion of the cases of phthisis that I have examined, pleuritic adhesions could be clearly and unmistakably made out.† Frequently, too, a history could be obtained dating the time of the pleuritis, and showing that it was the cause of the deterioration of health precipitating consumption. I believe these facts have not been sufficiently recognized

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* A paper on Pleuritis was read by Dr. Leaming before the New York Academy of Medicine, March 17, 1870, which was not printed. On the 7th of April the following paper was read, in which the leading points of the former paper are considered.

† In the post-mortem examinations recorded in the works of Laennec on the chest and of Louis on phthisis, pleuritic adhesions were found in the great majority of cases.

by the profession. I hope, that when attention is directed to this subject, we may be enabled to prevent such unhappy results.

It is plain that the more extensive the adhesions are, the more the lung will be bound down and crippled, and its capacity for vitalizing the blood will be diminished.

This variety of adhesions is the result, generally, of the most acute form of inflammation of the chest—pleuro-pneumonia. The prodromata are violent—there is great congestion, frequent pulse and high temperature. The friction sound of pleuritis is heard before the crepitant râle of pneumonia, and both precede dullness. This sthenic form of inflammation occurs mostly in hill countries, but is occasionally met with in cities, especially in children. It may be aborted by heroic treatment, taken in time, as by the sedative action of calomel.

*Case* I.—C. N., aged 3½ years, was taken sick August 12, 1869. On the 14th he became much worse, looked alarmingly ill, and the doctor was sent for. On the 15th the pulse was 138; respiration, 70; ratio, 1.9; temperature, 104½°. He was given calomel three grains, and tincture of aconite half a drop, with sweet spirits of nitre. On the 16th I saw him in consultation. Pulse, 120; respiration, 64; ratio, 1.8; temperature, 104. Physical signs: Friction murmur of the pleura; muffled respiratory murmurs, with commencing crepitant râles. The beginning of pleuro-pneumonia was recognized, and it was proposed to abort the disease. Eight grains of calomel were given at once, and one drop of the strong tincture of aconite root every hour till three drops were given, when all treatment was omitted. On the 17th—pulse, 98; respiration, 40; ratio, 2.4; temperature, 99. 18th—pulse, 100; respira-

tion, 32; ratio, 3.1; temperature, 98. The disease was aborted, no adhesions, no depression of vital power.

*Case* II.—A gentleman died in this city last November, not quite 60 years of age, gradually worn out with consumption. He was born at Great Barrington, Mass., where his early life was spent. All his family but himself were of notably robust constitutions, he only being thin and delicate. He dated his feeble health from the time of a violent inflammation of the chest when quite young. When I first saw him he stated that his chest disease was of long standing, and physical examination discovered phthisis advanced to the third stage in both lungs, with old adhesions in the lower part of the left lung, which were supposed at the time of the examination to have resulted from his childhood inflammation. It was believed to have been one of those cases where a predisposition to phthisis had been acquired from depressed vital power by crippling the action of the lung. The two cases were alike, I think, at their beginning; one was aborted, no damage remaining; the other passed through the course of the inflammation, and the consequence followed.*

Subacute pleuritis has its home in cities, or wherever there is a general lowering of vital power. Pleuro-pneumonia is an infrequent disease—that is, of the sthenic variety.†

Subacute pleuritis is of common occurrence; it frequently is without much pain, or none at all, and sometimes without the disturbing conditions which are usual. The effusion may pass off without damage,

*I should now classify this as a case of fibroid phthisis, third stage, for there were no cavities.

†Since this article was written, pleuro-pneumonia of an asthenic type has become common.

either from crippling adhesions or purulent change; and yet in every case there is danger that harm may result, and this danger is much increased by improper interference, and especially by the use of kidney-irritating diuretics, or plastic exudation-stimulating blisters.

I feel quite sure that adhesions of a vital depressing character may be the result of subacute pleuritis without treatment, but much more so when blisters have been actively used. Blisters in subacute pleuritis cause new adhesions of the same strong, binding character as those formed in the acute variety of pleuro-pneumonia.

Since the commencement of this discussion I have verified, as I think, more than one case of the damaging result of binding adhesions; but the following will suffice: A professional friend in the upper part of the city sent his servant to me for examination. The history was that she had subacute pleuritis with effusion some months ago. Before this her health was good; since, she has had cough, fever, and chills, with some night-sweats.

*Physical Examination.*—Left lung crippled by strong adhesions inferiorly and posteriorly, so that full expansion does not take place. The evidence of these adhesions is very plain and unmistakable. In the right lung, at the interscapular space, there are evidences of consolidation and active progress of tuberculization.* Effect following cause could hardly be better demonstrated than in this case; and yet it is not an exceptional one, for it was the frequent occurrence of just such cases that first drew my attention. I could multiply these instances, Mr. President; but I

*I would not now consider such physical signs evidence of tubercle, but of fibroid phthisis.

think it is sufficient to direct the attention of observers to this fact, in order that their experience should satisfy them of the truth of their position.

Adhesions that depress the vital power prevent motion of the lungs, and consequently must be in the middle or lower part of the pleura. We are thus enabled to make a clear distinction in our diagnosis. Pleuritic adhesions, intercurrent of phthisis, and which are conservative in their effect, always take place in the immediate vicinity of the active disease, and of course are mostly in the upper part of the lung. These are always limited by the extent of the tuberculosis; they enforce rest in the diseased part, check progress, and prevent perforation of the pleura. Conservative adhesions are in the upper part of the lung, damaging adhesions in the lower part; damaging adhesions take place before tubercular activity, conservative adhesions only after advanced phthisis.*

Tubercular deposits, which are hastened by the depression consequent upon binding adhesions, always appear in their natural place, that is, in the upper part of the lung, and quite frequently in the opposite lung.

I deem this subject important and not trivial; and if I am not much mistaken, the ground taken in the paper in respect to adhesions and active phthisis, as cause and effect, will be demonstrated by future unbiassed observers, and a recognition of the fact will enable us to apply the remedy, doing away with diuretics and blisters, which are damaging, and resorting to supporting and anti-tubercular remedies.

The next important question is, How shall we get rid of serum when it refuses to pass off in the natural

*My views with regard to adhesions and tuberculosis have been essentially modified by more extended experience, as will be seen in succeeding papers.

way? From the ground that I have already taken in regard to diuretics and blisters, I must reassert the opinion that their use in these cases should be abstained from altogether. The only proper method of removing the fluid innocuously, when Nature is unable to do it in her own way, is by the trochar and canula, in the method so ably demonstrated more than 250 times by our distinguished countryman, Dr. Bowditch, who honors us by his presence here to-night.

How soon after the effusion has taken place should it be removed? Nature, when not interfered with, removes in her own way all the fluid in from two to four weeks. The separation of the pleural surfaces, long enough to prevent adhesions, is conservative; but if this fluid remains three or four weeks, and shows no disposition to pass off, it should be removed by the canula. Dyspnœa or other urgent circumstances may make it necessary to do it much earlier, and then it may be only partially removed and conservative action still be observed. I have known a number of cases, where the fluid obstinately refused to pass off under the use of diuretics and blisters, speedily get well under the treatment of rhubarb and soda. One such occurred but lately, in a young lady in this city, Miss M., aged 16 years. I saw her on the 28th of January, 1870, in consultation: the left pleural cavity was filled with fluid, which resisted the usual treatment, and the heart was pressed far over to the right, and incapacity of the aortic valve could be made out in that position by the aortic obstructive systolic, and aortic diastolic regurgitant murmurs. Diuretics, etc., were stopped, and she was placed on rhubarb and soda. I saw her again on the 29th of March, in consultation, and learned from the attending physician that under this simple treatment

the effusion very soon passed away. Examination at this time showed that the lung had fully expanded, and was not damaged by adhesions.

In conclusion, Mr. President, the opinions and doctrines advanced in this paper may not harmonize entirely with those accepted by the profession; but I have only to say that they are the results of my sincere convictions, and are the result of some experience, and I place them before you for your deliberate judgment, with an earnest desire for the success of truth.

Dr. Peaslee said that Dr. L. wrote on the assumption of two propositions: first, that acute pleuritis is a serious disease; and second, that the occurrence of adhesions is a serious result. Though not claiming to be an expert, Dr. P. has seen much of pleuritis, and was surprised to hear it questioned whether acute pleuritis was serious, or whether adhesions were serious. He agrees with Dr. L. in the importance of the subject, otherwise we should have no interest in the paper. If the disease occurs we have three indications: subdue inflammation, get rid of the fluid, and prevent adhesion. And here it is important that we pay more attention to the conditions under which adhesions occur. It has been advanced that adhesions may occur without exudation; that they may be from proliferation of connective tissue, which, if true, and without inflammation, has nothing to do with the present topic. Confining ourselves to effusion and exudation, we must not forget that they are different events. Effusion is simply the pouring out of serum, while exudation is the pouring out of blood, minus the corpuscles. Effusion is not necessarily the event of inflammation; for we may have hydrothorax from disease of the heart, or from congestion and derangement of the liver, without pleuritis. We may also

have ascites with no peritonītis. Now there are four conditions under which adhesions occur. 1st, there must be, not effusion, but exudation; 2d, the exuded fluid must coagulate; 3d, the presence of epithelium; 4th, the two surfaces must be in contact. The reason that adhesions are less common in the lower part of the thorax is that the pleural surfaces are more constantly in motion by the action of the diaphragm in respiration.

In treatment, it is important that we make the distinction between acute pleuritis, as Dr. Leaming describes it, and effusion from other causes. In simple hydrothorax we need not fear adhesion, but the patient will suffer from compression. In actual pleuritis, where we have exudation, we may prevent adhesion by removing the fluid, if coagulation has not already commenced. He believes this will yet be done in orchitis and peritonitis. If the fluid be not removed, we may diminish the coagulability by administering calomel and alkaline remedies. He has no experience in blisters in the treatment of this disease, and never applies a blister to remove the cuticle; but whenever he applies one he removes it as soon as vesication has commenced, and applies a poultice. In this way he has never seen any harm result from their use.

## ARTICLE III.

### Respiratory Murmurs.*

Since the time of Laennec those engaged in investigating physical conditions of the chest have ever united in looking to the breath-sounds for the elementary key.

Able and distinguished men have given much of their lives to the consideration and practice of auscultation, but certainty in diagnosis in incipient disease is yet vainly desired. It must be that the method of study has been faulty, or that attention has been wrongly directed. Under these circumstances presumption may be pardoned, even if it should fail in the attempt to show a better way.

Laennec recognized both bronchial and pulmonary breath-sounds, and explained them as being caused by air-friction. In describing pulmonary respiration, he says: "On applying the cylinder, with its funnel-shaped cavity open, to the breast of a healthy person, we hear, during inspiration and expiration, a slight but extremely distinct murmur, answering to the entrance of the air into and expulsion from the air-cells of the lungs. This murmur may be compared to that produced by a pair of bellows whose valve makes no noise, or, still better, to that emitted by a person in a deep and placid sleep, who takes now and then a profound inspiration" (Forbes's Laennec, p. 29); and the translator adds in a foot-note: "It will be most easily and distinctively perceived by applying the naked ear to the chest of a child." Laennec's view is theoreti-

* Read before the Academy of Medicine, January 4, 1872.

cal, not based on a careful study of all the facts. Indeed, at that time the minute anatomy of the lung, and the constitution of the residual air, were not known. Subsequent opinions have been influenced more or less by Laennec's, especially in this, that all respiratory murmurs are considered to be air- and tube-friction sounds. Many differ from him as to the seat, but all agree with him as to the mechanism. M. Beau, of Paris, placed its seat in the pharynx; Dr. Sanderson, of Edinburgh, in the rima glottidis. Skoda, of Vienna, considered vesicular murmur as occurring only in inspiration, and being caused by air-friction, and he likened it to the noise one makes in forcing the air through the nearly-closed lips. He denies that the respiratory murmur has anything to do with the vesicular breathing, which, he says, is a purely bronchial sound. Andral called it a sound of pulmonary expansion or vesicular respiration, thus designating its seat, and giving it name.

Many speak of vesicular and respiratory murmurs as interchangeable terms. The late Dr. Hyde Salter placed the seat of the respiratory or vesicular murmur in the convective system, and mostly in the subpleural, minute bronchioli (*British and Foreign Med.-Chir. Rev.*, July, 1861). Dr. Waters, of Liverpool, whose prize essay on the minute anatomy of the human lung has done so much to increase our knowledge on this subject, describes the mode of connection of the bronchioli with the air-sacs. The opening sometimes is, as it were, a hole punched out, clean and round, and the air, passing in and out, must make a sound much in the same way as is done in a toy tin whistle. The late Dr. Cammann, of this city, believed the cause of the murmur to be the passage of air into the air-sacs and out again. Dr. Williams, after speak-

ing of portions of the chest where blowing sounds are heard, goes on to say: "Then there is the vesicular respiration, which is heard in most other parts of the chest; it is a diffused murmur caused by the air penetrating through the minutest tubes, and into their numerous vesicles or cells." Dr. Gerhard, of Philadelphia ("Lectures on the Diagnosis, Pathology, and Treatment of Diseases of the Chest"), says: "The sound of air entering the vesicles is different from that caused by its passage through the tubes, and the former is, therefore, known as the vesicular sound, the latter as the tubal or blowing sound. The vesicular sound is often called a murmur, from its softness and diffusion over a large space, and cannot be produced unless the vesicles are healthy or nearly so." And again he says the cause of difference "seems to be the different manner in which the air impinges upon the vesicles and tubes. But the vesicular sound is in part owing to the vibration of the air, and in part to the noise produced by the dilating of the vesicles themselves." *

Dr. Walshe represents the natural respiratory murmurs as caused by inspiration and expiration, for which there is usually a healthy type, "commonly termed—*a*, pulmonary or vesicular; *b*, bronchial; *c*, tracheal; *d*, laryngeal; *e*, pharyngeal, according to the part of the respiratory apparatus from which the sounds audible externally are transmitted." Dr. Corrigan divides the sounds heard in auscultation into "simple sounds or murmurs, and compound sounds or rattles. . . . All the sounds heard in the chest belong to one or the other of those two kinds; and if, when you hear a

* Dr. Gerhard's explanation shows that he very nearly comprehended the dual character of the respiratory murmurs.

sound, the exact nature of which you may be in doubt of, you will first refer it to its class, your labor in determining what it is will be very much diminished." The American editor of "Stokes on the Chest" describes vesicular murmur as that "of a soft and gentle, or, as it has been otherwise described, a mellow, continuous, gradually-developed, breezy murmur, unattended with a sensation either of dryness or humidity; and we are properly cautioned by M. Fournet and his reviewer not to expect a character of sound which conveys the notion of a successive dilatation of separate vesicles, or, as it is sometimes called, pure and vesicular." Dr. Hyde Salter says: "There is another reason, to which I have not referred, which makes me think that the respiratory murmur must have a tubular or *quasi* tubular seat, and cannot be formed in the air-cells; it is, that fine crepitation, such as that of pneumonia, *supplants* it; it does not merely drown it, it supplants it; the two do not coexist;" and farther on: "If, then, pneumonic crepitation is a veritable tube-sound, and its seat the microscopical tubes immediately subtending the air-cells, the supplanting and destruction of the respiratory murmur by it would show that this latter has an identical seat, and is therefore a tube-sound." This explains Dr. Salter's views as to the seat and cause of the murmur. He believes it to be caused by the passage of air through these microscopic air-tubes, just before they reach the vesicles; and, as he is one of the latest and most brilliant writers on this subject, perhaps he represents the more advanced views of the profession. He does not deny that sounds formed anywhere in the convective system, from the mouth or nose to the smaller bronchi, mingle with and enter into the composition of the respiratory murmur, but he denies that the air-vesicles or

alveoli have anything to do in forming the sound. He believes the sound is formed in the bronchioli, immediately subtending the pulmonary pleura.

Carefully examining the opinions of all these different writers, it is evident that some consider the respiratory murmur as having a single seat and cause, while others recognize its composite character. I am not aware that any one has heretofore ever attempted to analyze the murmur, and study its constituents separately as well as together. They speak of the vesicular character, the pulmonary quality of the respiration, but they attempt no analysis. To show that this may, and ought to be done, in order to attain unto a higher grade of excellence in diagnosis, is the main object of this paper. A clear understanding of this whole matter will make it necessary, as preliminary, to look at the minute anatomy of the tissue of the lungs, and of the bronchial system; secondly, the circulation of the lungs and of the bronchial system; and, thirdly, the characteristics and constitution of the residual air, its object and office.

The bronchial or convective system may be called the broncho-respiratory system, and the pulmonary the true respiratory system. They differ in almost every respect. The office of the broncho-respiratory is to convey air into the true respiratory system, while the true respiratory system is where the great function of oxygenating or of vitalizing the blood is perfected. The bronchial system is characterized by cartilage in its fibrous sheath. In the upper part, where it is necessary to prevent collapse of the tubes, the cartilage is in nearly perfect rings, but as the tubes pass into the lung-structure, which is occupied by residual air, the cartilage gradually loses the character of rings, and appears merely as deposits occurring at irregular in-

tervals, down so far as the bronchial arteries extend, to where the bronchial veins commence to carry back the blood that has passed through the capillaries of the bronchial mucous membrane. The mucous membrane also of the broncho-respiratory system, is different from that of the true respiratory system in this, that it is ciliated epithelial mucous membrane, while the other is of tesselated basement epithelium.* The circulation also is entirely different. The convective system is supplied by the bronchial arteries alone; the pulmonary substance by both the pulmonary artery, and the nutrient arteries of the lungs, which are the connecting link between the two systems. The nutritive arteries arise from the bronchial arteries, but have no accompanying veins. Thus, blood, after performing the proper office of nutrition in the pulmonary tissue, is at once reaërated, and passes into the venous radicles of the pulmonary vein prepared for systemic circulation.†

The bronchial arteries have been called the nutritive arteries by anatomists, but they have not dwelt upon the fact that the *venæ comites* do not attend these arteries into the pulmonary structure, and that, consequently, this gives them a peculiar character. The bronchial veins return all the blood of the bronchial arteries; the nutritive arteries are derived from the bronchial but have no veins. Their blood is reaërated where they do their work, and it finds its way into the venous radicles of the pulmonary vein as arterial

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* The ciliated columnar epithelium, so characteristic of the bronchial mucous membrane, ceases at the commencement of the alveoli. (Dr. Waters "On the Chest," 1868.

† Ibid., pp. 16, 17. Also, Stricker's "Histology," 1872, p. 443. Niemeyer, vol. i. p. 60; Wilson's "Anatomy," p. 514, 1859; Gray's "Anatomy," p. 720.

blood. This anomaly in the circulation is of great interest in explaining physiological causes and pathological effects. In pneumonia it is the nutrient artery, accompanied with its plexes of ganglia of the organic nerve, lymphatics, etc., that preserves the life of the part, and governs the whole process of resolution. We can all remember the anxiety of practitioners, in the past, to prevent abscess and gangrene of the lung after inflammation. But time, and a more careful study of the natural history of the disease, have proved to us that gangrene and abscess are rare accidents, even when no treatment at all is had. This peculiar arrangement of the nutrient artery gives us an early knowledge, in many cases, of commencing phthisis. Occupation of the air-sacs by tubercle interferes with the circulation, and blood is thrown back upon the bronchial artery, and the result is bronchorrhagia, a conservative act; for, like the application of leeches, it sets the absorbents actively at work to remove the cause—the new tubercle. And in this way cases of early phthisis are self-cured, or, at all events, ameliorated, and the physician is guided in his treatment.

This singular fact in the circulation was discovered by the late Dr. Cammann, in making his experiments to prove the non-anastomosis of the arteries of the lung. Using a colored fluid suitable for fine injections, he found that, when he injected the pulmonary artery, the fluid returned easily by the pulmonary vein; but, injecting the pulmonary vein, the fluid not only passed into the pulmonary artery, but, if the injection was carefully continued, it would also find its way into the bronchial arteries. Then, again injecting the bronchial arteries, he found that the fluid after a little time passed into the pulmonary vein; this proved that there was communication between the bronchial arteries and the

pulmonary vein, but not with the pulmonary artery.* This was shortly after 1840, and before, I believe, any experiments had been made in Europe, in regard to this circulation. Since then, several observers have come to nearly the same conclusion. Drs. Williams and Adriani believe "the vessels of the bronchial mucous membrane terminate in the pulmonary veins, and those of the deeper plexus in the bronchial veins." Dr. Waters says, after explaining his experiments, which were very full and minute: "That a distinct and free communication exists between the bronchial vessels and the pulmonary veins admits of ocular proof. I have seen, with the aid of the dissecting microscope, the small vessels passing from the outer surface of the bronchial tubes, and forming a small trunk, which terminated in a pulmonary vein." Dr. Waters also says:† "It may be said that such a view militates against the generally-received opinion of the purity of the blood returned to the left side of the heart, for, if the bronchial blood is poured into the pulmonary veins, it is returned to the left auricle without undergoing the process of aëration. I would answer that the view I have taken is supported by anatomical facts, a basis on which all physiological theories should be founded." I remember that Dr. Cammann, also, could not reconcile the incongruity of the apparent fact that venous blood passed directly with the aërated blood of the pulmonary vein, and into the left heart. Both of these gentlemen overlooked the truth that the blood from the nutrient artery passes through capillaries in the true respiratory system on its way to the radicles of the pulmonary

* Communicated to me by Dr. Cammann.

† "Minute Anatomy of the Human Lungs."

vein, and, of course, is reaërated. Dr. Robert Lee, if my memory serves me (for I have not the paper at hand), says that the extension of the bronchial artery, after it has quit company with the vein, receives additions from the mammary and intercostal arteries, and has the proper title of nutrient artery. I do not quote his words, but the substance, as I remember it.

I believe, then, I am warranted in holding that there is a complete difference in the blood vessels of the convective and of the pulmonary systems. The nutrient arteries of the bronchial system have their *venæ comites;* the nutrient arteries of the true respiratory system have no accompanying veins, but pass their blood reaërated directly into the pulmonary vein, prepared for systemic circulation. The nutrient artery is no exception to the rule of complete difference in the two systems, for in its office it belongs wholly to the true respiratory. The vessels of the bronchial system are the bronchial arteries and veins; the vessels of the true respiratory are the pulmonary artery and vein, and the nutrient artery of the lungs passes from one into the other.

Where the bronchial system ends the pulmonary begins, and the division is sufficiently marked—it is where cartilage ceases and alveoli commence. The structure of the true respiratory system is composed of terminal bronchii, in which are developed alveoli and the air-sacs, that is, wherever alveoli are found. Its whole object or office is aëration of the blood of the body. It is greatly distensible, and in this differs from the convective system, which is but little so, and its formation evidences design in the economy of space and for its especial purpose. The bronchioli have alveoli developed in their sides, but not to the same extent as in the air-sacs, which are but a skeleton net-

work for the convenient spreading out of alveoli, with their *rete mirabile* of capillaries, for the aëration of the blood. The terminal bronchus enlarges at its end, and the air-sacs are developed from this enlargement, according to Dr. Waters, as a cluster of leaves are, sometimes, from the end of a twig. From six to thirteen of these air-sacs are in connection with the enlarged end of a terminal bronchus, and this little cluster forms a lobulette—a complete type of the whole lung. Each lobulette has its terminal bronchus and air-sacs for the development of alveoli, its twig of pulmonary artery and vein, its branch of nutrient artery, with the accompanying gangliæ of organic nerve, lacteals, absorbents, etc. A collection of lobulettes forms a lobule, and a number of these constitute a lobe. The fibrous bands of the bronchial sheath, both the white and the yellow, are continued, though with great tenuity, through the terminal bronchi into the air-sacs. They surround the mouth of each air-sac, and give firmness to the frame of each alveolus. Muscular fibres also accompany these bands, though their presence is doubted on account of their extreme tenuity. Niemeyer speaks of muscular fibres as present in the true respiratory system. In emphysema, the air-sacs lose their power of contraction, and become dilated, causing great suffering and disability to the patient. Time and freedom from catarrh allow the function of contraction, which is a muscular habit, to return.

Physiologists describe residual air as filling the respiratory system as high up as the third or fourth division of the bronchi. It not only fills the true respiratory system, but distends it. The elements of the distending force are: atmospheric pressure, muscular contraction, rarefaction, the laws of diffusion of gases, and that of affinitive attraction between oxygen and

venous blood. The residual air occupies its position with such persistence as to be with difficulty dislodged after death, even with much pressure. It keeps its place with vastly greater tenacity, during life, when each element of force is in active operation.

During inspiration, the contraction of the diaphragm increases the capacity of the chest, and at the same time the epiglottis is raised, and the weight of the atmosphere operates actively in dilating the lungs. Rarefaction of the newly-inspired air takes place upon inspiration, owing to its immediate and intimate admixture with the residual air, and is the third element of dilating force. The residual air is estimated to be 170 cubic inches, and the inspired air at 20. At each inspiration, therefore, the residual air will be increased about one-tenth in dilating power, *plus* the rarefaction of the inspired air.

But the peculiar elements of this expanding force are, the laws of the diffusion of gases, and of affinitive attraction between the unaërated blood-globules, in the capillaries of the *rete mirabile* of the alveoli, and the oxygen, equally distributed throughout the residual air. Chemistry demonstrates that gases differently constituted in certain relations instantly intermix when brought together. The inspired air and the residual air present these differences. Air entering the convective system moves in a body through the bronchial tubes till it meets the residual air, when, the law of the diffusion of gases operating, immediate admixture takes place. The residual air is instantly renewed with oxygen, in accordance with this law. The inspired atmospheric air moves through the convective system, as far as the fourth division of the bronchiæ, with no other resistance than the friction of the tubes. When it meets the residual air, it is immediately con-

sumed, as it were, and does not accumulate, causing resistance. On this account the inspired air moves with increasing velocity, producing air and tube-friction murmur. Tidal air in health is only heard in inspiration. Velocity of the moving air-friction in the tube is the cause of murmur. Any one may demonstrate this fact by breathing through a tube gently, when there will be no murmur; but, if he increase the velocity of the moving air, he will get sound, which will be increased in sonority and raised in pitch just in accordance with the rate of motion. In health, in unconscious breathing, expiration is not heard, and we know by experience that, when it is heard in unconscious breathing, there is disease; it may be phthisis, or it may be emphysema—other conditions must determine which. A harsh murmur may be produced at will, by hurrying the respiration. It is heard in systemic diseases like cholera, or in diseases of particular organs, as in cardiac apnœa, or Bright's small kidney. The cause of murmur, in air moving in a tube, no matter what are the other conditions, or the disease, is the *velocity*, increasing the air and tube-friction.

Prof. John W. Draper has given a convincing explanation, based on accurate experimentation of affinitive attraction in the systemic capillaries, as one of the efficient causes of the circulation. The same power operates in the pulmonic circulation, but with this important addition, that the attraction is not alone in the pulmonic tissues and the blood, but principally in the venous blood and the oxygen of the residual air. This is the cause that brings the venous blood and oxygen together, in order that the blood may be purified and fitted to continue the life of the body. Let us endeavor to comprehend the intricate mechanism

of the respiratory act. Inspiration has taken place—twenty cubic inches have been added to the residual air, evenly and equally admixed—dilatation has taken place with force, and is continued and increased by the rarefaction of heat. The true respiratory system, by its muscular power, contracts forcibly, antagonizing the dilating residual air. Each particle of pure air, acknowledging its attraction for the venous blood, presses up to the alveolus, through the struggling mass, and rushes to the blood-globule in the capillary —makes the interchange—gives up its oxygen, and receives in return tissues, detritus, and carbonaceous materials, loses its attraction, becomes passive, then is crowded back by other eager particles pressing forward, until finally it finds itself well up in the bronchus, with its filthy load, whence it is expired. The blood-globule from the pulmonary artery, entering the capillary of the alveolus, hurries along through the *rete mirabile*, drawn by its affinity for oxygen, till it meets a particle of pure air, makes the interchange, loses its activity, but is now pushed onward by other globules pressing forward from behind, till it finds itself in the venous radicle of the pulmonary vein, fitted for systemic circulation.

The movement of the blood-globules is much assisted by the contraction and relaxation of the muscular fibres of the true respiratory system. Different bundles of these fibres, contracting and relaxing in succession, give not only a living vibratory motion, which assists in hurrying the globules along, but produce a susurrus, which, being heard at the chest-wall in multitudinous concert, with consonating tube and air-friction sound is true respiratory murmur. These facts in minute anatomy and physiology (and they hardly admit of any dispute) prove that the residual air, as a

body, has no more motion than has the bottom of the deep sea. No change can occur except molecular, and none other is necessary. The law of diffusion of gases assures the comparative purity of the residual air, as well as its constant and guarded impurity, which is so necessary for the accomplishment of the vital act.* The circulation would not go on if each blood-globule should immediately come in contact with pure air, for then it would lose its impelling force, and, all of the globules alike losing their attraction, there would be stasis. Instead of this, both in the blood and the residual air, each globule and each air-particle moves in perfect order, never in each other's way. This shows how the individual may live in bad air for a time, resisting its evil tendencies, and even that of poisonous gases. It shows also why medical inhalations fail in their object. Medicated vapors have little or no admission into the residual air. Even oxygen gas, which is sometimes serviceable, can only supply atmospheric deficiencies. It can neither do the harm nor the good that has been predicated for it. An animal may even live for a time in pure oxygen gas, the active interchange taking place between the gas and the blood re-

* "This diffusion [of gases] is constantly going on, so that the air in the pulmonary vesicles, where the interchange of gases with the blood takes place, maintains a pretty uniform composition. The process of aëration of the blood, therefore, has none of the intermittent character which attends the mechanical processes of respiration." Flint, Physiology, vol. i. p. 407.

"Now it is obvious if no provision existed for mingling the air inspired with the air already occupying the lungs, the former would penetrate no further than the larger air-passages. The change must be attributed to the 'mutual diffusion' of gases." Carpenter, Physiology, Phila., 1853. See, also, Kirke, Physiology, p. 235. Cyclopædia of Anat. and Phys., Lond., 1847-49, vol. iv., part i., p. 362.

storing the necessary grade of impurity in the residual air.

If, then, the only change or motion that is possible in the residual air be molecular, what becomes of the theories of air and tube-friction murmurs, whether in the terminal bronchioli or the air-sacs and alveoli, as cause of the so-called vesicular murmur? Are they not physical impossibilities? And, too, what becomes of the theories of the mechanism of crepitant *râle?* If there is no motion but the molecular, there can be no bursting of bubbles in the microscopic tubes, and that theory falls. If the residual air constantly and forcibly distends the true respiratory system, how can the walls of bronchioli and air-sacs come together, to be separated again by each inspiration of fresh air, so as to produce fine crepitant râle? This theory, likewise, supposes a physical impossibility. All theories, whether of vesicular murmur or crepitant râle, which ignore the presence of the residual air, are of necessity incompetent. The fact that residual air has none but molecular motion may be demonstrated by a distensible bag, as of India-rubber. While it is being forcibly filled with air, there will be air and tube-friction murmur at the mouth only, where the air moves in a body with velocity. The body of air in the bag will be increased by particles of air sliding in among each other and without sound. But there will be resisting vibratory sound in the walls of the tense dilating bag; different, however, from that of the contracting true respiratory murmur in this, that it is only heard during dilatation, while the other is continuous, because owing to active muscular contraction. Dr. Hyde Salter says, after speaking of the occupancy of the true respiratory system by residual air, and that about twenty cubic inches of atmospheric air are added at each inspiration:

"Each air-cell is, therefore, a tenth larger at inspiration than at expiration. Now, it is inconceivable that this slight variation in the capacity of these shallow open concavities should be attended with any sound. I cannot conceive it possible. For, be it remembered that the air-cells are not nearly-closed cavities communicating by constricted orifices with the general cavity of the lobular passage, but wide-mouthed and patulous like a teacup.* And be it remembered, too, that in respiration the air is not pumped out of and into the cells, but, as they undergo this slight change of volume, a small part of their contents passes just without them, and then again, on their recovering their capacity, from without just within them, if one can speak of 'within' and 'without,' in reference to such slight interchange of situation. For, really, the renovation of the air in the tissues of the lung does not depend on its actual removal, but upon the law of the diffusion of gases."

This reasoning is cogent and unanswerable. It proves beyond cavil that there is no motion in the air-sacs and alveoli to produce air and tube-friction sound, and yet he attempts to show that there is such motion in the terminal bronchioli and intralobular passages. He says: "But while the movement of the air at each alveolus would be so slight, so almost inappreciable, the collective expansion of all the alveoli common to a lobular passage, and the consequent abstraction of air from the general cavity, would be considerable, and would create a considerable rush of air into the lobular

* Dr. Salter refers to the alveoli which are developed both in the bronchioli and in the air-sacs. The alveoli developed within the air-sacs, although wide mouthed and patulous, are yet enclosed in sacs which have only a narrow opening, preventing movements of air *en masse*.

passage to supply its place, for the modicum of air, however small, appropriated by each dilating air-cell, would of course be multiplied by the number of cells communicating with the common axial cavity of the lobular passage."

Dr. Salter's able reasoning shows that there is not enough motion in the alveoli or air-sacs to cause sound, and it is strange that he did not see that the same reasoning applies with equal force to the air in the bronchioles and intralobular passages. The residual air occupies these passages just as well as it does the air-sacs; one-tenth is added at each inspiration to the whole body of residual air, and Dr. Salter himself has said that these small bronchial tubes were largely distensible; consequently, the velocity of motion in these passages where alveoli are developed must be too little, if there be any at all, to produce any sound. There certainly can be no rush; indeed, I have already shown that there can be no motion, except the molecular. But, for argument's sake, if there should be motion in these minute tubes, as Dr. Salter claims, it could not possibly have the velocity necessary to cause sound. Dr. Salter's argument to prove that the seat of crepitant râle and the seat of respiratory murmur are the same—"The râle supplants the true respiratory murmur; the two do not coexist"—heretofore quoted, is convincing. Had he placed the seat in the air-sacs and alveoli as well as in the terminal bronchioli, he would have been correct, for then he must have acknowledged that it could not be by tube and air-friction, and he would have been forced to accept the true explanation, that of dilatation and contraction. Crepitant râle indicates the commencement of the process of inflammation, and it supplants the true respiratory murmur.

Let us study the evidence in the light of the true respiratory murmur.

If you have lately examined the chest of a person in health, and have noted the murmur in its fullness and perfection, and should be called to see the same person suffering from a chill, with pain in the head, back, limbs, etc., and should again examine the respiration carefully, you will still hear the true respiratory murmur, but it will be obscured or muffled. All the capillaries of the lung are crowded with blood, and this is the explanation of the muffled murmur. If you wait a few hours, and again examine him, you find the true respiratory murmur absent, and, in place of it, the fine crepitant râle. The congestion of the capillaries of the lung still remains; there is scarcely a perceptible difference in the percussion-note; the residual air still occupies its seat in the true respiratory system, and it still continues to dilate the air-sacs, alveoli, and terminal tubes. Whatever change has taken place must have been at the seat of the true respiratory murmur.

In tissues that may be seen, what is the first result of inflammation? Is it not that plastic material is thrown out into the connective tissue? This, also, must take place in the lungs. The connective tissue of the lungs, delicate as it is, has been filled with plastic material. It has become thickened and stiffened; it cannot contract, and the true respiratory murmur is gone; but it must yield, though unwillingly, to the dilating force of the residual air, increased one-tenth at each inspiration, separating newly-formed plastic exudations, causing sound, which we hear as fine crackling, and call it crepitant râle. If we wait a few hours more, and examine again, we will find that crepitant râle as well as true respiratory murmur has gone, and all is silent, or there may be bronchial or

tubular breathing. Exudation has been poured into the true respiratory system, and consolidation is the result. The seat of crepitant râle is now become the seat of exudation.

This is the true mechanism of crepitant râle. In this paper I have endeavored to show that the bronchial respiratory system is entirely different from the true respiratory system in anatomy, physiology, object, and use, and that the physical signs of pathological change are equally distinct and different. That the residual air, occupying, as it does, the true respiratory system with force, precludes the idea of currents of air within the lungs, and consequently the accepted theories of the vesicular or respiratory murmurs and of the formation of crepitant râle are necessarily incompetent. If my points are well taken, and the proof convincing, the profession will eventually sustain the truth, and much that has been received as settled literature will be swept away as rubbish, to give room for truer and better grounds of faith.

The composite character of the respiratory murmur must be made evident, analytically as well as synthetically. The two elements, different in cause, character, and seat, must be individually studied in order that we may correctly understand their significance in pathological changes. We may present their union and the result to the eye, thus:

| | |
|---|---|
| Broncho-respiratory murmur. | Respiratory murmur. |
| True respiratory murmur. | |

The reasons for introducing a new terminology are, that broncho respiratory and true respiratory are descriptive, and indicate the seat of the murmurs. The term vesicular murmur was applied by Andral, sup-

posing that it described the minute anatomy of the seat of the murmur.

Later investigations show that the term is misapplied, for there are no structures that may properly be called vesicles in the lungs. Again, the terms vesicular and respiratory have been applied indiscriminately, and their present use would lead to confusion and misapprehension.

In order to practically study these murmurs, it will best be done by selecting a healthy person about twenty-five years of age, with perfectly-developed chest and with muscles not hardened by manual labor.

### RESPIRATORY MURMURS.

Place the ear lightly yet firmly to the chest, allow the head to rise and fall with the respiration, listen to the breath-sounds of the patient, and breathe with him synchronously. The tidal-air of bronchial murmur will first catch the ear modified by the true respiratory murmur, and, as has been described, it is like the sighing of the trees over our heads in the forest, when the leaves are gently stirred by the breeze. The character and quality of the respiratory murmur depend upon the absence or excess of one or the other of the composing elements. If the true respiratory murmur be maximum in fullness, the tidal air-sound will be short, only heard in inspiration, and will be of the soft, breezy character described as gently sighing.

While, if the broncho-respiratory be in excess, the tidal-air sound will be harsh, raised in pitch, and will be heard both in inspiration and expiration, and becomes a sign of disease as the other is of health.

### BRONCHO-RESPIRATORY MURMUR.

Broncho-respiratory murmur may be studied by

forcing the breathing, when it will be heard in both inspiration and expiration, and its harshness, loudness, and pitch will depend upon the force given to the respiration. This murmur may be heard in its natural perfection in the chest of a child, before the true respiratory murmur has been developed.

### TRUE RESPIRATORY MURMUR.

The ear accustomed to auscultation, after a few moments of concentration of the attention upon the respiratory murmur, will recognize its dual composition thus: if the chest be perfect in condition, the tidal-air sound will be heard in inspiration only—soft and short, like breathing gently through the closed teeth—while the true respiratory murmur will be continuous, increasing in fullness by inspiration and diminishing in expiration. It is of low pitch, and is like the roaring of the sea at a distance, the waves breaking on an even shore of sand; or, better still, like the sound made by bees in cold weather, when the hive is tapped with the finger. It is like the innumerable vibrations of the wings of bees, increasing to maximum by full inspiration like the coming waves on the sea-shore, and decreasing in expiration as they recede. If the breath be held, this murmur may be heard without admixture, for there can then be no bronchial friction murmur. The sound is the susurrus of the delicate muscular fibres of the true respiratory system, contracting and relaxing over the dilating and resisting compressed residual air. If the breath be held after a full *inspiration*, the murmur will be at its maximum; if it be held after *expiration*, it will be at its minimum fullness. It cannot be exaggerated, as has been said of the so-called vesicular murmur. If the true respiratory system be unduly dilated, it loses its power to contract on the

residual air, and the murmur wholly ceases. This is a sign of emphysema, and is proof of the muscular cause or origin of the sound which may return again after rest.

This murmur only commences to be developed in the child at eight years of age, becomes recognizable at twelve, but is only fully developed at maturity. A beginner in auscultation may recognize true respiratory murmur in a good subject with ease. But, when the chest has lost its excellent quality as an acoustic chamber by physical changes, resulting from inflammation, or when, from disease of the lung itself, the natural respiratory murmur has been altered or lost, or when the chest, although in its natural conditions, may be covered by thick and hardened muscles, the trained, expert ear only can arrive at diagnostic truth.

These facts, instead of being a matter of discouragement, should induce beginners to pursue auscultation with untiring assiduity, knowing that the end will crown them as masters in physical diagnosis. The ability to recognize true respiratory murmur under any conditions, to analyze its quality, and measure its power, gives its possessor the means of knowing even the approach of that most insidious disease, phthisis, and suggests the method of prevention.

The true respiratory system, air-sacs, alveoli, nutrient artery, ganglia of the organic nervous system, with absorbents, etc., all require active use for the prevention of disease. Phthisis does not begin in the lower part of the lungs, which are constantly and actively in motion. If we insure the same kind of exercise in the upper part, we prevent and may even arrest incipient disease.

## ARTICLE IV.

### Plastic Exudation Within the Pleura.

*Dry Pleurisy.**

It is the known experience of all who make autopsies that thickened pleuræ and pleuritic adhesions, the results of plastic exudation, are of frequent occurrence; and yet the text-books and teachers of physical diagnosis give us no signs for their easy and ready recognition. Practitioners who have watched cases all through a whole course of illness, ending fatally, have been surprised at the post-mortems to find abundant evidence of plastic exudation within the pleura, although none had been suspected during life. It seems strange that medical observers in the past should have been content not to have recognized physical signs of such obvious pathological conditions. One standard writer, however, acknowledges the existence of intra-pleural noises. Dr. Walshe arranges and classifies them ("On Diseases of the Lungs," p. 113), and his arrangement is evidently intended to cover all possible intra-pleural sounds. But the world of medicine has given him credit for hardly more than hypothetical reasoning. Recently, one whose experience is ample, and whose reputation is world-wide, said in substance that it was the general voice of the profession that pleuritic adhesions were not recognizable by signs, and that if they were the knowledge would be of no practical value. This undoubtedly represents the accepted views of the leaders of medical thought. In regard to

* Read before the N. Y. State Medical Society at Albany, Feb. 5th, 1873.

the possibility of intra-pleural noises, Dr. Stokes says: "It is only when the surfaces are rendered dry by an arrest of secretion, or roughened by the effusion of lymph, that their motions produce sound perceptible to the ear." Dr. Walshe says, speaking of the normal conditions of healthy pleura: "This noiselessness of movement of the pleural surfaces upon each other depends at once upon their perfect smoothness and slight humidity." The silent movement of the healthy pleura upon itself, and the rhonchoid effect of plastic exudation, as quoted from Drs. Walshe and Stokes, and which are self-evident in their truthfulness, seem not to have had their proper influence upon the minds of medical observers, and this may be the reason why these conditions producing sound have been so generally overlooked. I believe the explanation is simply that it is because the common signs belonging to plastic exudation within the pleura have been misinterpreted. Mucous râles have been considered, and truly, as being caused by mucus moving in the larger bronchi; and also the so-called subcrepitant or sub-mucous râles, by a parity of reasoning, have been described as being formed in the smaller tubes, the size of the tube proportioning the size of the râle. From the first postulate, only partially true in itself, all the succeeding errors have arisen. Mucous râles may be formed in the larger bronchi; but subcrepitant râles cannot occur in the bronchioli. I think I have demonstrated in a former paper (*N. Y. Medical Journal*, May, 1872) that currents of air in the true respiratory system are impossible. All of the true respiratory system—that is, all of that part of the lungs which is beyond the third and fourth divisions of the bronchi—is filled persistently by the residual air. No motion can ever occur there, such as could produce friction murmurs.

There is no motion within the residual air of any force or velocity in any direction—no movement at all except the molecular, which is silent, and there is no need of any other. Therefore, there being no air movement, there can be no subcrepitant râles formed within the true respiratory system, for their mechanism in the bronchioli becomes a physical impossibility.

Above the third or fourth divisions of the bronchi there is tidal air, producing air friction murmur; and if mucus be present that may be moved along with it, producing râles or rhonchi which may be heard at the chest wall. Their mechanism places them at different distances from the ear, and the same râle may be heard at several points, a fact that is noted by that delicate organ with marvellous accuracy; while plastic râles formed within the pleura are always near the ear, moving *with* the lung within the parietes in expansion and contraction of the chest, and are heard only over the site of their occurrence. The true mechanism of subcrepitant râles is always intra-pleural, and depends upon the presence of exuded lymph. But true mucous râles may be telephoned into the chest-wall at long distances from their occurrence, and yet are easily recognized as such.

Convincing proof of the correctness of these novel positions may be had by a careful comparison of clinical notes of the *site* of râles with their post-mortem revelations. In my own experience, both in hospital and private practice, I have never failed to find the presence of plastic exudation after death as cause of the râles heard during life. If the plastic matter be of recent formation, the signs will be soft-tearing râles, like tearing moist flannel, two or three threads at a time. The râles may be exceedingly delicate, and require close attention and acute hearing to recognize

them, and a corresponding condition of the exudation will appear at the autopsy. Again, the adhesions may be months or years old, and the râles will be correspondingly hard and dry, sometimes creaking like new leather, and the plastic matter will be found after death dense and strong, like bands of cartilage. Dr. Walshe is in error when he says: "We cannot predicate from the character of the friction sound the state of the pleural exudation," for, availing ourselves of the *real significance* of subcrepitant râles, we may diagnosticate with sufficient accuracy the condition of the exudation. The character of the râles denotes the age of the adhesions. When tubercular deposits take place near the surface, pleuritic exudation may be an accompaniment. Consequently these râles, although intrapleural, have been considered, and in some cases the diagnosis would be correct, as evidence of softening and breaking down of tuberculous masses, or caseous degenerations. Still, these signs, *always* interpreted as pulmonary, may lead to error of diagnosis, for the râles may disappear, as every observer knows, and no lesion of lung structure remain, for pleural inflammation may take place, even at the apex, and no tuberculosis be present.

Those who never diagnosticate phthisis unless they hear moist râles will mostly make their diagnosis too late for any benefit to the patient, and sometimes, possibly, diagnosticate disease which does not exist. Perhaps plastic exudation within the pleura and tuberculosis in the lung, when recent, may both be absorbed and no lesion result. Yet, plastic râles heard at the upper part of the lung are a grave sign, and should engage our earnest and immediate attention, so frequently are they the precursors of disorganization. They may be considered a warning by which, if

heeded, a disastrous result may be avoided. But when disintegration of lung *does* take place, the râles remain and increase in loudness and dryness, and, when the walls of the cavity are indurated, they are reverberated and crackling in character. This was called tubercular crackling by Laennec and his followers, although the mechanism was acknowledged to be a mystery. If, however, any one will carefully note the site of these signs during life, and their correspondence with pathological conditions after death, he can hardly fail to be convinced that the cause of the râles is in the stretching of inter-pleural adhesions near or over a cavity. But if the walls of the cavity are soft and yielding, and, more so, if they contain fluid, the sounds produced by the stretching adhesions will be liquid in character, like gurgling, but may be distinguished from the true by being persistent, while the true gurgling disappears when the cavity is emptied.

Dr. Walshe relates a case (p. 116, "On Diseases of the Lungs") which prove that these crackling râles may be formed outside of the lung. He says: "An extremely abundant, medium-sized rhonchus occurring almost in puffs, and having the liquid, bubbling character in a most marked manner, was day after day, during the week previous to death, detected in the entire height of the left side posteriorly. The explanation of the rhonchus naturally suggesting itself was that it depended upon œdema of the pulmonary tissue generally. At the post-mortem examination, however, I found this explanation was inadmissible, for the thin lamella of tissue between the cavity and the surface was as hard as cartilage, and contained not a particle of serosity; nor was the organ in any part distinctly infiltrated with fluid, being on the contrary particularly dry,

from its excessive induration." He afterwards says: "Subsequent experience has amply proved the correctness of this explanation, and shown that moist sounds, rhonchoid in properties, are producible whenever adventitious tissue within the pleura is infiltrated with serosity and the movements of the chest continue free."

Proof that these crackling râles are formed outside the lung is, that when the plastic exudation has been so abundant as to bind down a large space of lung, so as to prevent *all* motion in the pleura, there will be no râles. The disintegrated lung underneath being in the same condition, both when the pleura is moveable and when it is not, the râles should be the same in each were they formed within the lung.

The following cases are offered as proof that subcrepitant râles always have an intra-pleural origin:

*Case* I.—Margaret Simpson, New York, 21, single. Examined at my office, September 23d, 1870, for admisson into the House of Rest for Consumptives, at Tremont, N. Y. Right side, clavicular and mammary regions; loss of true respiratory murmur; broncho respiratory prolonged in expiration, and raised in pitch in inspiration. Dullness with raised pitch on percussion. Dry adhesion râles, subcrepitant in character, at the lower portion of the lung, both before and behind, but more extensively behind. Left apex—a large cavity recognized by cavernous respiration, with dry crackling reverberations in the cavity. Dullness and raised pitch over the whole upper region of the left lung, with subcrepitant râles at the lower part, before and behind. Died October 15th, 1870. Post-mortem by H. M. Sprague, M.D., physician to the institution. Large cavity in the upper part of the left lung, and cavernules below. Cavernules in the upper part of

the right lung. *Both lungs completely bound to the chest wall with pleuritic adhesions.*

*Case* II.—Rebecca Robinson, born in Ireland, age 35. Examined at my office October 29th, 1870, for admission into the House of Rest, etc. Right lung—evidence of tuberculosis; second stage, in upper part. Left lung—large cavity in the upper part; signs of pleuritic adhesions (subcrepitant râles) in the lower part of both lungs—more in the left than in the right. Post-mortem examination December 18th, 1870, by H. M. Sprague, M.D., physician to the institution. Large cavity found in the left lung, smaller in the right; *both pleura bound firmly to the chest wall by adhesions.*

*Case* III.—Mrs. P., aged 60, examined by H. M. Sprague, M.D., for admission to the House of Rest for Consumptives, Tremont, N. Y., November 25th, 1872. Respiratory murmur altered at the left apex; almost entirely absent. The so-called subcrepitant rhonchus heard over both lungs, anteriorly and posteriorly. December 10th, complained of having taken cold; coughed rather more than usual; was feverish. The pleuritic friction râles (subcrepitant) much more marked than before. Died the next day. Autopsy twenty-four hours after death. Right lung tuberculous; is the seat of pneumonia; commencement of second stage. Pleura thickened at the upper part from plastic exudation. *Lung bound to the chest wall over its entire surface.* Adhesions infiltrated with serosity. *Left lung bound to the chest wall firmly in every part,* though separated from it by a layer of serum. Over the apex the pleura thickened to about one fourth of an inch, and hard as leather. Left lung very much congested at apex. Dilated bronchus of the size of the little finger. The inner surface ragged and ulcer-

ated, and the walls thickened and fibrous. About half a pint of serum remained in each pleural cavity after removing the lungs.

*Case* IV.—W. M——, aged 35. Admitted January 6th, 1873, to the House of Rest for Consumptives. Examined by H. M. Sprague, M.D. Cavity in the left apex and subcrepitant râles heard over the whole of both lungs. On the day of admission, complained of having taken cold, was chilly during the day; two subsequent days remained in about the same condition, but grew worse and died January 9th, the third day after admission. Autopsy twenty-four hours after death. *Both lungs bound down firmly to the chest wall*, and adhesions infiltrated with serum. About half a pint of serum in each pleural cavity, after removing the lungs. A large cavity in the upper lobe of the left lung, containing a small half ounce of purulent matter. The cavity was lined with a diphtheritic membrane, easily removed. The pleura over the left apex very much thickened; lower lobes congested and studded with grayish, fibrous nodules, of the size of bird-shot—(tubercles?). The right lung was the seat of acute pneumonia. Middle lobe, near the root of the lung, contained a few of the same fibrous nodules, which were interspersed with caseous nodules of the size of a pea.

Evidently the post-mortem revelations in Dr. Walshe's case were a surprise, so contrary were they to the usual interpretation of signs. Still, holding to the pulmonary origin of subcrepitant râles generally, he gives the following directions for making differential diagnoses! "The crackling form, in itself indistinguishable from some conditions of subcrepitant rhonchus, may be diagnosticated by the existence of friction sounds, constant or occasional, and by its being

unaffected by coughing. Mere moisture in the plastic matter within the pleura seems enough to give a rhonchoid character to friction sounds." I am quite confident that if Dr. Walshe might test all the subcrepitant râles by comparing them with the post-mortem conditions, as he did in the case of crackling, he would be equally convinced that they all have an intra-pleural origin. Dr. Sprague's cases prove not only that the crackling râles, but also the subcrepitant, are of intra-pleural origin. They also prove that sounds that have been dry may become liquid in character, when infiltrated with serosity from accession of disease. Old adhesions, when the patient is well, may become almost silent, and escape ordinary attention, but if the patient take cold they become again loud and distinct. The soft-tearing râles, spoken of before, indicate recent exudation, and the softer and more liquid the sound, the more recent.

Commencing at this point in the scale, we may rise through all the gradations to dry crackling and creaking of old adhesions, which are as unyielding as cartilage. The age of the exudation may thus be pretty accurately determined, and the knowledge prove of great practical value. Recent deposits of lymph may be entirely reabsorbed, leaving the lung free in its movement afterward. Long observation convinces me that plastic exudation and reabsorption (without medicine) are a common occurrence, the power of the vigorous life of the body being sufficient to remove effusion or exudation within the cavities, unaided. But if the power of the life, the organic life, be not sufficient, aid must be given by the intelligent physician, or the exudation remains, doing more or less damage, by binding the lung more or less firmly. Unwise intermeddling is more to be feared even than

unaided and insufficient organic life, because it reduces still farther the already weakened vital power. As such unwise intermeddling I would specify long-continued depressing medical agents and confinement in impure air.

If the disease be mistaken for bronchitis, which is not unusual when recent, and if the patient be kept in a warm room, in impure air, and dosed with nauseating and depressing expectorants, the vital power may be so depressed as to be unable to remove the exudation, and a crippling of the lung will be the consequence. In all cases assistance will be most efficacious in the earliest stages; then, if ever, antiplastic remedies are serviceable. In extreme cases, those of exceptional violence, or when the amount or extent of exudation is excessive, the powerfully sedative action of calomel may abort the disease so completely that not a vestige of it will remain—this, too, without any draught upon the life-power of the individual. Twenty, thirty, forty, or even sixty grains, placed on the tongue, may be necessary to produce this sedative action. No one but the physician attending can judge of the dose proper to the case. The proper action of the calomel will simply be the disappearance of the grave signs and symptoms. The heart's action will be more regular, fuller, and slower. The plastic exudation will rapidly disappear by reabsorption. There will be no purging, no ptyalism, and no exhaustion of vital power. I know of nothing so satisfactory in medicine as the proper application of this powerful remedy, when given in the disease needing it, and at the right time. The dose should be given so as not to be repeated—strike but once—repeated dosing may do harm. In milder forms of the disease, alkalies, especially muriate of ammonia, may do the work safely, but more slowly.

One thing should not be forgotten, and that is the anti-plastic effect of pure air and simple food. These several means, adapted to each individual case, will seldom fail to cause the absorption of plastic exudation when recent, but it is almost impossible, if not quite so, to hasten absorption in old and cartilaginous adhesions. The most that can be done for them is to remedy their progressing contraction, and obviate their depressing and tubercular tendencies. This may be done by systematically expanding the chest, endeavoring to elongate the adhesions and to increase the vital capacity. Recent adhesions may with certainty be rendered innocuous by expanding the chest, even though they be not immediately reabsorbed.

In the etiology of tuberculosis writers have considered every other cause but that of plastic exudation. This has no place. But I firmly believe that when the true signification of subcrepitant râles shall be known as plastic râles, all will agree with me that this cause is far more potential than all the rest.

Professor Austin Flint, Sr., in a late able paper on Etiology of Phthisis, adverts to the fact which Niemeyer and his followers lay so much stress upon—that in many of the cases of phthisis the patients date the commencement of their illness from a cold. Niemeyer claims this as proof of the catarrhal origin of the disease. Every clinical observer must have been struck with the fact that some patients are positive as to the time and particulars of their attack, such as irregular chills, dry cough, sometimes accompanied with viscid expectoration, etc.; also, that others are just as positive that their decline commenced without cough or other occult symptoms. They had weariness, loss of appetite, loss of flesh, and dyspepsia, long before the cough characteristic of this disease commenced. If

we take pains to number them, we shall find that there are about two-thirds who *date* the time of their commencing illness, to one-third who cannot; and that this is about the proportion of plastic exudation preceding and accompanying phthisis, and that of idiopathic tuberculosis. In a paper on Pleuritis, which I had the honor to read before the New York Academy of Medicine three or four years ago, I expressed the opinion that a large majority of the cases of phthisis which had been under my care, at the class of chest diseases at the Demilt Dispensary, gave evidence by physical signs, frequently confirmed by the history, that the disease had commenced with pleurisy—meaning plastic adhesions. I could not have been clear in my language, for I was generally misunderstood. Nearly every one who discussed my paper considered effusion of serum as a necessary accompaniment of pleurisy; consequently my conclusions were disputed. To make this matter clear, I will now state that I did not, nor do I now, assert that I have any evidence that pleurisy with effusion has an especial tendency to end in phthisis. On the contrary, I wish to repeat what I said then, that the effusion is conservative, preventing the evil tendencies of a lung crippled by adhesions, for the effusion separates the pleura till the danger is past.

Cases of phthisis may and do follow effusion when not removed in due time, but they are rare, and I fully agree with those gentlemen who so stoutly opposed views I did not hold. But I did hold, and do now, with more conviction than ever, that plastic exudation, crippling the lung, has a depressing tendency upon the organic life of the body, and is very frequently followed by phthisis.

The intelligent observer who, having the evidence of exudation râles before him, shall follow the cases to

the dead-house, will have proof that adhesions have a powerful influence in precipitating phthisis. Plastic exudation within the pleura obeys the same law which it does in other parts of the body. It continues to contract some time after its organization. Consequently the lung becomes more and more crippled, and it must either decline into desuetude, provoking tuberculosis, or in its efforts to free itself become emphysematous. It is the laocoon of animal life struggling within the tightening folds of the plastic python. Emphysema is the opposite of tuberculosis, and systematic forcible expansion of the crippled lung may produce temporary emphysema, effectually preventing phthisis.

My private note-book shows that more than two-thirds of the cases of phthisis examined in my office have had plastic exudation within the pleura, which must have influenced its commencement or progress. Sometimes phthisis follows immediately, or rather begins with the exudation, and then it is frequently accompanied by hæmoptysis, which should not be interfered with; it is nature's self-preserving act. In the early stages of fibroid phthisis hæmoptysis may occur from time to time, after each new exudation of plastic matter; but eventually the bleeding ceases from extension of fibroid in the lung, and to great emaciation from the diminished quantity of circulating blood.

Hæmoptysis in fibroid phthisis is an encouraging sign, for it is evidence that nature has not given up the fight.

The following cases, taken from many others, are given to illustrate the depressing effects of plastic exudation within the pleura, and also its tubercular tendencies, and that these tendencies are preventable:

*Case* I.—A gentleman about 32 years old had

hæmoptysis in 1861, and the left lung remained with physical signs of arrested phthisis. In October, 1872, while in the country, he had hemorrhages, lasting about one week. He was said to have had pneumonia also. In December he returned to the city, and examination detected plastic exudation signs over the whole of the right pleura, and that extensive disorganization had taken place in the right lung. Plastic exudation, no doubt, took place at the time of the hæmoptysis in October, more than two months before. Disintegration of the lung must have commenced immediately after the plastic exudation, for he was examined a few days before going into the country, and at that time the right lung was free from disease. It is possible that a powerfully sedative dose of calomel, placed on his tongue at the time of the hæmoptysis, might have relieved the crippled lung, and have saved the patient's life.

*Case* II.—In the early part of July, 1872, a professional gentleman was found to have plastic exudation in the left pleura, extending from below up to the internal angle of the scapula. The subcrepitant râles were abundant, but confined to these limits. True respiratory murmur could be heard over the rest of both lungs. He had been complaining for a few weeks of irregular chills and cough, but thought these symptoms due to malaria. About the first week in August he was again examined. The adhesion râles remained as at the first examination, but both lungs had lost expansion, and also true respiratory murmur.

He went to the country and returned to the city again in October, being absent about nine weeks. It was found that he had a large cavity near the root of the lung. The right lung was without expansion, and without true respiratory murmur, but there was no

consolidation. Plastic exudation ensued in the right lung in December, precipitating the end.

In this case the plastic exudation preceded the phthisis a definite period. There were *no* signs of tubercle in July. There *were* signs of tubercle in both lungs in August, and rapid disorganization of the left lung followed.

*Case* III.—Mrs. —— had plastic exudation about three years ago, and signs of adhesions remained in the right pleura at the lower part. She had been examined by several physicians, who had heard the adhesion râles, and also the true respiratory murmur in both lungs. In August, 1872, she had chills and cough, followed by loss of flesh and strength, and some spitting of blood. In October she was carefully examined, and it was found she had lost chest expansion and true respiratory murmur, and that there were soft subcrepitant râles over both lungs, though not abundant.

She was directed to systematically expand the lungs, to be much in the open air, to take plentifully of milk and farinaceous food; cod-liver oil; also muriate of ammonia, quinine, and iron. She followed the advice thoroughly, and in four weeks' time she was again examined, when there was better expansion of the chest, and true respiratory murmur could again be heard. In December she had recovered her usual health.

I cannot doubt that in this case phthisis was prevented.

*Case* IV.*—February 19th, 1873. I. H——, a square-built, heavy-chested man, came to my office for examination; he had been spitting blood at times during

* Added since the paper was read at Albany.

the eight days previous. At two or three different times he spat up about half a pint of blood, and at others a smaller amount. His wife had been ill several months with consumption, and he had been much with her, and was frightened at the idea that he might have caught the disease. He was very pale, and had a coated tongue. Examination detected abundant plastic exudation râles in the lower part of the right pleura, forming a band about three inches broad, extending from the vertebra around to the junction of the ribs with the cartilages. The lungs were slightly emphysematous, but otherwise healthy. Believing this to be a case which warranted an attempt at perfect relief by forcing absorption of the exuded matter, I wrote for one scruple of calomel and ten grains of sugar, and directed him to place the powder on the back part of his tongue that evening. February 22d he called again to say the hæmoptysis had ceased, that he had had two or three full passages from the bowels, and that he felt well. Examination showed the lungs to be free in movement and without râles of any kind.

This case may be considered as evidence: 1st. That the sedative action of calomel may cause rapid absorption of plastic exudation within the pleura; 2d. That intra-pleural plastic exudation may cause hæmoptysis, with great depressing effect upon the ganglionic life; and that the liability to tuberculosis from these conditions, especially when there is phthisical proclivity, is obvious.

I could adduce many other cases illustrating all these points, were it necessary, and time sufficient, but content myself with believing that the means of diagnosis are within the available reach of all, and that convincing proofs will constantly occur to the most skeptical.

I will therefore merely restate the points which I consider to be the most important in connection with the subject.

1st. Adhesions and thickened pleura are among the most frequent pathological results discovered at autopsies.

2d. That generally they are not known during life, as the sounds they make are considered pulmonary, and errors of diagnosis and errors of treatment are the consequence.

3d. Plastic exudation within the pleura is even a more frequent accident than can be determined at autopsies; in esoric cases reabsorption taking place so speedily that no adhesions remain.

4th. Binding adhesions prevent expansion of the chest, and consequently of the true respiratory system, hurry the heart-beat, derange the digestive organs, prevent proper assimilation of food, depress the vital force, and, unless emphysema results, precipitate phthisis pulmonalis.

5th. About two thirds of the cases of phthisis seen in clinical practice commence with or after adhesions; that hæmoptysis frequently is coexistent, and that such cases are remediable.

6th. That the remedial means are systematic but not forcible expansion of the lungs, change of air, a proper supply of food that may be easily assimilated; and that medicines, when used, should be antiplastic and tonic, sustaining the organic life.

## ARTICLE V.

### Physical Signs of Inter-pleural Pathological Processes.*

In health the pleuræ are smooth, opposing surfaces, free in motion, and lubricated by their natural secretion. They cover the inner costal wall, the outer surface of the pericardium, nearly the entire upper surface of the diaphragm, and all the surfaces of the lungs. They help to form the mediastinum, and surround the origin of the great vessels and of the air-passages. In short, they line the great acoustic chamber of the chest, and cover the sound-producing organs which it contains.

The acoustic properties of the normal chest are so perfect that the most delicate signs, such as true respiratory murmur or aortic regurgitation, are delivered through its walls to the ear without loss or change. The healthy pleuræ are no obstacle to the free passage of sound and at the same time are no cause of sound in themselves. It is like looking into an open room filled with light.

But by inflammatory process they cease to be silent themselves, and modify or prevent sound passing through them. It is like looking into a room filled with cloud, through obscured glass; nothing is clearly seen.

The pleuræ are prone to disease changes. Mental depression, physical exhaustion, or sudden alternations of temperature, may cause hyperæmia, and plastic exudation. The change is a vital process, but indicates

* *The Medical Record*, May 25, 1878.

a depressed condition of organic life. It occurs in cellular tissue and on serous surfaces—very frequently on the pleural. Comparatively few autopsies are made without discovering more or less of inter-pleural thickening and adhesions.

"Physicians of old did not regard them as preternatural; nor do many at the present day consider them as necessarily connected with inflammation. This opinion is founded upon the fact of these adhesions being met with in individuals not known to have suffered from any inflammatory affection of the chest. But until satisfactorily traced to some other cause, it would appear more proper to refer these exclusively to an inflammatory origin." (Hasse's "Pathological Anatomy," p. 182.) The process may be summarized as follows:

First, local vital exhaustion, vaso-motor paresis, stasis, hyperæmia; then the white globules, "the wandering amœba," pass through the meshes of the walls of the capillaries, and, unless immediately absorbed, organize and result in adhesions and thickened pleura. This may take place without rise of temperature. Can it be called inflammation?

More or less of impaired health follows, with obscure symptoms, and periodicity in rise and fall of temperature. New exudations take place from time to time, crippling the respiratory organs, and seriously implicating the circulation, until the patient dies, worn out, with resulting complications of all the vital organs.

These serious pathological results have hitherto been unrecognized, except in part and inadequately, during life by physical signs. In March, 1870, I had the honor of reading a paper before the New York Academy of Medicine on Pleuritis. In the discussion which followed, Dr. Flint, Sr., remarked that he

was "not aware that there are any distinctive physical signs of permanent adhesions that can be depended upon as pathognomonic." This was undoubtedly then, and perhaps is largely yet, the received opinion of the profession, Dr. Walshe alone, among the authorities, interpreting certain physical signs as of adhesions and pleural thickening. I had adopted the generally received opinions; but clinical experience, obtained in the class of chest diseases at Demilt Dispensary, caused me to question their truth. There were repeated occurrences which seemed to me to furnish incontrovertible proof that many of the physical signs of the chest had been misinterpreted. But even yet I was not prepared to give up my preconceived idea of the local origin of mucous and crepitant râles.

But about ten years since, a patient came into St. Luke's Hospital, from Bellevue, with a disputed diagnosis. The case had been affirmed to be one of simple hydrothorax, and then again to be hydropneumothorax. Both opinions were correct, paradoxical as it may seem, for upon examination it was found that on the right side there was dullness and loss of respiratory murmur up to about three inches above the diaphragm; and bordering the upper line of dullness, and encircling the lung, there was crepitus and subcrepitus. It was simple circumscribed hydrothorax. But upon directing the patient to take a forced inspiration and hold the breath, air was forced into the artificial chamber made by the adhesions, and the case was immediately changed into one of hydropneumothorax. In a little while the air escaped, and the case was as at first, hydrothorax. To account for this it was necessary to suppose that there was a valvular opening, through which air could come from the lung. The fluid was removed by intercostal incision, and the case

kept under observation. After a time the left side showed signs of disease. There were mucous râles and gurgles, and progressive loss of weight and strength. The case was frequently referred to as an example of tubercular phthisis. The râles were of various sizes, and were considered as signs of tubercular infiltration and honey-combed cavernules.

At the autopsy the circumscribing adhesions, with the valvular opening in the lung, were found on the right side, as was expected; but, to our astonishment, there was no structural change in the left lung. Between the pleural surfaces, however, there was a large amount of plastic exudation, together with a small quantity of viscid fluid; at many points also there were firm adhesions. These inter-pleural deposits were evidently the only source of the sounds I had misinterpreted as signs of the tubercle and tuberculous cavities of small size. I was convinced that the same conditions had frequently deceived me in other cases.

Since then I have, in repeated instances, carefully noted and recorded the locality of râles and their distinctive characteristics, for the purpose of testing them in relation to pathological conditions, to be revealed by autopsies. In no instance have I have found them to disagree with the interpretation that their cause lay in an inter-pleural process.

The following case is one in which old, firm, and close inter-pleural adhesions drew the heart upward, and caused murmurs by displacement.

*Case* I.—J. S. T., an honored member of our profession, called on me in company with Dr. Otis, in the spring of 1876. About seven years before he had pleuro-pneumonia, and several times since slight attacks of pleurisy. He was short of breath, and distressed after exertion, or on going up stairs; he had

at times severe pain in the region of the heart like angina. Aneurism of the aorta and valvular disease of the heart were feared and had been diagnosticated.

Examination discovered some dullness at the summits of the lungs. There was flat-wooden percussion note over both. There was very little expansion, and the movement of the chest was restricted. There were a few râles of various sizes over the greater part of the chest. At the lower angle of the scapula of the left side there were no râles, nor any movement of the lung even in forced inspiration, but coughing produced short, fine crepitus immediately under the ear.

True-respiratory murmur was feeble generally, but absent at the apices of both lungs. The apex beat of the heart was between the fourth and fifth ribs, a little to the left, and there was a systolic murmur.

Diagnosis: old extensive adhesions in both pleural cavities, drawing upwards the heart and lungs. There is no disease of the heart or of the arteries.

On the 12th of October, 1876, he received a wound, to the right of the sternum over the auricle, by a piece of a brass tube imbedding itself in the lung and the pericardium. Pericarditis and pneumonia followed, and he died on the 20th.

The day before his death, and probably for some time previously, there were abundant soft "mucous" râles. These were diagnosticated also as interpleural. The autopsy revealed pericarditis and pneumonia of the whole of the right lung, which was consolidated—the mould of the ribs remaining on its surface after it was removed from the chest.

Hence, no air could have entered the lung. New exudation had taken place among the old adhesions, and efforts at respiration moved the chest-wall over the solid lung, thus producing "mucous râles."

*Case* II—Is kindly given in a letter from Prof. J. L. Little, and is equally decisive: "My dear Doctor, I cheerfully comply with your request that I would furnish you with the points in the history of a case bearing on the subject of your paper. I was called to see a patient in consultation with Dr. Roëdiger, on August 14th last, and found a man about forty-five years of age, who was suffering slight pain in the left side—no cough, no expectoration, high temperature and frequent feeble pulse. On auscultation, subcrepitant râles could be heard on the posterior surface of the left side of the chest. These were more abundant and of a much coarser quality at the upper part of the lung, although more or less subcrepitation could be heard from apex to base. On percussion, flatness was discovered over the entire upper portion of lung; the lower showed but slight dullness. I saw the patient in consultation on the 17th, 18th, and 19th. At the last visit, eighteen hours before death, subcrepitation was heard, as at first examination. On forced expansion after coughing, the râles were markedly increased in number, and seemed to be very near the ear. Patient died August 20th. Autopsy by Drs. Roëdiger and Nesbitt. Left lung was found solid with pneumonia, except the lower part of the inferior lobe. The upper was in a state of gray hepatization, the middle red. The lower part was very much congested, but crepitated on pressure. The pleuræ were covered with plastic exudation, but the adhesions were slight. The false membrane covering the upper third of the lung was three or four millimetres in thickness. The lower portion was covered with only a thin layer. In this case, Doctor, the râles heard over the posterior surface of the chest were without doubt due to the exutionda on the surface of the pleuræ. No air could possibly

have entered the upper or middle portions of the lung for some days before death.

"Yours truly, J. L. LITTLE."

These two cases are evidence of a positive character verified by post-mortem examination.

*Case* III.*—C. M——, saleswoman, eighteen years old, came to my office for examination on the 14th of September, 1877. Percussion-note dull over lower part of left lung in front and up to the middle of interscapular space behind. There was bronchophony, bronchial breathing with subcrepitant râles. At the lower portion of the lung in front was *fine crepitus.* As the lung was consolidated by pneumonia no air could enter it, and consequently the râles must have been in the pleuræ. This evidence is further corroborated by the fact that since then the pneumonia has cleared up, resonance returning, but subcrepitant râles remain in place of crepitant.

These three cases may be regarded not as unusual, but as typical, and they furnish proof: 1st, That "mucous" (so called), 2d, that subcrepitant, 3d, that crepitant râles may all have their local origin within the pleuræ.

It is difficult to overcome preconceived opinions even with evidence perfectly conclusive to an unprejudiced mind. Still the facts which I have given, and others which I shall further relate, must commend the subject to all candid observers.

Has the generally received opinion that all large, soft, moist râles are caused by bursting bubbles in the

* This case is of a class common to all practitioners, and is introduced as such. It has no novelty, but after the post-mortem evidence of Cases I. and II. the clinical evidence of consolidated lung becomes proof of the impossibility of the râles being interpulmonary.

bronchi ever been put to the test of careful experiment? On the contrary, is it not the general experience that abundant mucous râles may exist without expectoration, and profuse expectoration without râles, or only with such as are distant from the ear, and which disappear upon expectoration? Has not the received opinion that all the râles, of whatever size, liquidity or dryness, have their origin in the lungs, and that the size of the bronchia determines the size of the râle, been adopted by pupil from teacher, from the time of Laennec to the present, without regard to the obvious fact that the large râles are most frequently heard over portions of the chest where the bronchial tubes are very small, and the small râles where they are large?

Early auscultators explained the respiratory murmurs of health, as well as the rhonchi of disease, as being formed by the air passing through the bronchi into the vesicles and out again; that the friction of this body of air in motion caused vesicular murmur and bronchial breathing; and that should mucus collect, it would be moved along bursting bubbles in its way—crepitant, subcrepitant and mucous, according to the size of the râle. Later, another theory was proposed, and by many adopted, which still regarded the size of the tube as governing the size of the râles. According to this theory, the tube being lined with adhesive mucus, collapsed after expiration, and the sides cohering, inspiration would again force them apart, causing râles. Neither theory recognizes obstruction to the free passage of air into the air-sacs and out again, yet the residual air certainly occupies the true respiratory system and does not admit air moving in a body. The tidal air physiologists estimate to be about one-tenth part of that in the lungs. So

that after expiration there still remain nine-tenths, occupying the true respiratory system. This is the residual air. When inspiration again takes place the column of *in*-moving air passes in a body to about the third or fourth division of the bronchia, and can go no further, but mixes with the residual air, obeying the law of the diffusion of gases.

It is evident, in view of these facts, that both theories are impossible. The existence of so large a mass of residual air in the air-cells and smaller bronchial tubes, and also the existence of consolidated lung tissue (in which solid material fills the spaces previously occupied by the residual air), both show conclusively that all râles called crepitant and subcrepitant, when heard under these conditions are not intra-pulmonary, and that "mucous râles," when not clearly traceable to the large bronchi, are also not intra-bronchial, and consequently all râles not clearly traceable to the larger air-passages are inter-pleural.

Mucus in the upper bronchia may cause true *mucous râles*, which are intermittent. The mucus accumulates, the râles are heard; it is expectorated, and they are gone. In suffocative catarrh, and in approaching dissolution, the râles are continuous.

It would seem possible that fibroid lung could also produce subcrepitation in cases where the lung is adherent to the chest-wall. But of this I have no proof.

The following case of fibroid phthisis was characterized by a variety of râles and rhonchi, which would deceive any one who did not recognize them as signs of an inter-pleural pathological process. They were very suggestive of cavernulous phthisis and of disease of the heart.

*Case* IV.—W. L——, about sixty years of age, merchant, while in Scotland in 1874, had pneumonia, and

since then had had frequent colds, causing short, spasmodic cough, with gradually increasing dyspnœa. He came under my care early in 1876. His breath was short and hurried. There were râles over both lungs; in some places coarse and rattling, and in others smaller, even fine crepitus. At the lower part of the right inter-scapular space, and below the scapula, they were coarse, moist, and gurgling. Under the right axilla down to the diaphragm there were creaking as well as dry râles. Under the left clavicle there were mucous gurgles, and under the left scapula there were fine crepitant and subcrepitant dry râles. There was general flatness under percussion, with raised pitch over most of the chest. There was an audible systolic murmur at the apex beat. The impulse was felt almost as high as the nipple, and there was also impulse in the second interspace.

The diagnosis was extensive plastic exudation between the pleural surfaces, forming adhesions which had drawn the lungs and heart upwards. The dyspnœa and cardiac complications were the consequence of these changes, and there was no other serious lesion. At first he improved under treatment, and gained more than an inch in chest expansion, and was able to get about with much less difficulty than before treatment, but in May an attack of pneumonia increased the amount of plastic exudation, and he lost more by subsequent contraction than he had previously gained in expansion. During the summer, in the country, he was under the immediate care of Dr. Ely, Newburg. He was able at times to ride out, but new attacks of exudation lessened his vital capacity, and finally, after another "cold," he had increased disease, from which he died Oct. 8, 1876.

*Autopsy* by Prof. Delafield, Oct. 10th. Present, Drs.

Jones, Dudley, G. A. Peters, Ely, and Leaming.—"Body much emaciated, cadaverous discoloration already evident on abdomen. *Pericardium* contains a little serum. Apex of heart on level with lower edge of fourth rib—distant three and one-fourth inches from median line. Upper border of heart on level with lower edge of first rib. Long axis of heart turned somewhat in vertical direction.

"*Lungs.*—Left side, very extensive old adhesions covering the entire lung. Left lung, upper lobe at the apex, some bands and patches of pigmental fibrous tissue. Lower lobe, lower third, bands of new fibrous tissue and red hepatization—the red hepatization is recent. Right lung, old adhesions over entire lung. Upper lobe, the same diffuse fibrous tissue, but more abundant.

"*Heart.*—Right ventricle contains large yellow post-mortem clot. Pulmonary valves a little thickened at their attached edges. Ventricle a little dilated, walls of normal thickness, tricuspid valve a little thickened.

"*Left ventricle* contains a small post-mortem clot. Cavity rather diminished. Walls normal thickness.

"*Aortic valves* somewhat atheromatous and stiffened, and on ventricular aspect of one leaf a small fibrous projection. Mitral valve a little thickened and atheromatous.

"*Kidneys*—Normal size, capsule not adherent, surface smooth, cortex normal in appearance and thickness. Aorta markedly atheromatous.

"(Signed) FRANCIS DELAFIELD."

This instructive case illustrates the diagnostic value of correctly locating the site of râles and rhonchi. Interpreted, as inter-pleural they indicate old cellular adhesion with fluid in the interstices, extending

into the lungs—fibroid phthisis—which was proved to be correct.

The first step in these complicated pathological changes was plastic exudation between the pleuræ, which, becoming organized, formed adhesions, and these in turn gave rise to all the subsequent diseased conditions in the lungs and of the heart.

*Case* V.—M. M., æt. 40, single. Saw her in consultation with Dr. E. D. Hudson, Jr., September 7, 1877, morning. Heart and great vessels gave no evidence of disease. Pulse and cardiac sounds were feeble and frequent, suggesting fatty degeneration. Chest expansion was not more than half an inch; respiratory murmur very faint; very little air entering the lungs. No signs of disease of the lungs or pleuræ were discovered. But there was evident obstruction in the air-passages, the patient gasping for breath. Lung free from dullness. Highest local pitch in respiration traced to the larynx, and the obstruction was believed to be at this point.

The laryngoscope, in the skilled hands of Dr. Lefferts, proved this opinion to be erroneous.

Evening.—Consultation with Drs. Hudson and Lincoln. There was now found, at the summit of the left lung, perceptible dullness and flatness under percussion, and soft, tearing râles in auscultation, conditions which had developed since morning. Respiration was more difficult, and during the examination became so great that unconsciousness resulted. No time was to be lost, and Dr. Lincoln performed tracheotomy, and the obstruction was found to be below the trachea. No evidence of aneurism was discovered. Dr. G. F. Shrady informs me that he refused to give this patient ether for an operation previously, because he suspected aneurism. Death occurred early on the morning of the 8th,

*Autopsy*, afternoon of the same day, by Dr. Hudson, in the presence of Drs. Lefferts, Hitchcock, and Kemp. "Cause of death, aneurism at the posterior surface of arch of aorta descending. Trachea and bronchia atrophied by the pressure of the tumor. Heart fatty, lungs reduced in volume, but normal otherwise. At the left apex the opposed pleural surfaces were agglutinated, the soft adhesions offering slight resistance in separating. Several older, organized but elastic adhesions spanned the left pleural cavity." (Notes of the autopsy kindly furnished by Dr. Hudson.)

This case is evidence that plastic exudation may be diagnosticated as soon as it takes place. There were neither râles nor dullness in the morning, but there were both in the evening, and fresh plastic material was found at the autopsy. Hasse says: "The first appearance of inflammation of the pleura consists in a congested state of its blood-vessels, which are seen congregated here and there, in dense though delicate nets, beneath the still transparent membrane. At certain points the bright-red color deepens and becomes more equalized; these points are somewhat prominent, and, though scattered at first, presently crowd together and get encompassed with a progressively enlarging zone of gorged blood-vessels. At the same time patches and streaks are observed either darker than the rest, and not unlike little ecchymoses, or else of a pale red hue, as if from imbibition. The pleura now speedily loses its smoothness and polish, becoming dull, and looking, as Laennec expresses it, as if daubed over with a paint-brush. This redness gradually spreads until in most instances the whole, says 'Gendrin,' becomes uniform.

"The first rudiments of an adventitious membrane now become perceptible, the spots originally reddened,

and that chiefly by repletion of the vessels, presenting little dull white or yellowish points which rise above the serous surface in the shape of flat granules, and ultimately coalesce." (Hasse's "Pathological Anatomy," p. 133.)

All of the pathological changes described above, from the first congested blood-vessels in nets to the final covering of the whole pleura with lymph, produce the following signs: First, muffling; second, alterations of the respiratory murmur; and then, finally, râles and rhonchi, indicating exudation of plastic material. Every step of the pathological process is characterized by its appropriate physical signs.

Experience and a nice education of the ear make an early diagnosis easy and certain, and enable the practitioner to use remedies which, if employed in good season, remove the disability and the danger.

*Case* VI.—(Plastic signs removed by hygiene.)—F. J., about 26 years of age, while at business in Wall Street, in 1874, suddenly began to raise blood, and came immediately to my office. There was an area over the right scapula, where soft, tearing râles could be heard, and there was also flatness under percussion. He was advised to take a walking expedition of two or three weeks' duration. This he did, and returned in health, not a vestige of the plastic râles remaining; nor has he had any return of chest signs or symptoms since.

It is possible that had he remained at his exhausting business under all the depressing influences which had produced their conditions, his lowered vitality would have been still farther depressed, and his case would have resulted in phthisis, as many others have done—so important is it to connect physical signs correctly with their true pathology.

Plastic exudation upon the pulmonary pleural sur-

face has the immediate effect of obstructing the capillary circulation in that part of the true respiratory system which subtends the deposit. If it is not quickly reabsorbed it becomes organized, and contracts, causing still greater obstruction. Hæmoptysis frequently results—it may be immediately, but in most cases not until after two or three weeks after the exudation or even longer.

The reason of this is evident, if we consider the minute anatomy of the circulation of the true respiratory system. The nutrient arteries of the lungs are derived principally from the bronchial, and differ from all others in the body, in the fact that they have no returning veins; no *venæ comites*. The nutrient capillaries, after performing their special function, anastomose with the radicles of the pulmonary vein, and their blood is reaërated even while performing its office, and hence, notwithstanding this apparent anomaly, arterial blood is alone forced into the left heart.

Consequently obstruction of the nutrient capillaries throws their blood back upon the bronchial arteries, which might seriously interfere with the circulation, except for a provision of nature, by which mucus is exuded copiously through the mucous membrane (bronchorrhœa), or perhaps blood (bronchorrhagia). So that either may be an important symptom of plastic exudation, and if carefully sought for the plastic râles will be found.

*Case* VII.—G. B., a distinguished surgeon, April 1, 1876, had pneumonic sputa; pulse 100, temperature 100°. Had some oppression in breathing, but no pain. Auscultation discovered no râles on either side. True respiratory murmur was everywhere good, except over a part of the middle lobe of the right lung—a space about as large as the palm of the hand—where there

were also perceptible dullness and raised pitch. Diagnosis: Centric pneumonia of the middle lobe of the right lung. The next day the pulse was 70 and the temperature 97°. Sputa the same as the day before, and so it remained on the 3d and on the 4th. Subsequently the temperature was as low as 93°.

On the night of the 5th of April he suffered great dyspnœa, and auscultation discovered abundant râles of crepitant and subcrepitant size, covering that part of the middle lobe of the right lung, posteriorly, over which true respiratory murmur was absent at the first examination on the 1st of April. The explanation is that the centric pneumonia had extended to the pleural surface, plastic exudation had joined the pleuræ together, and crepitant râle and bronchial breathing were plainly heard. The dyspnœa fom which he suffered was the consequence of these adhesions.

He gradually improved until the 27th of April, when he went to Fortress Monroe for change of air. Shortly afterwards he had a return of dyspnœa, and as it increased he came home on the 5th of May. He now had moist, tearing râles low down on the *left side.* The heart was restrained in motion, and the first sound was altered in character. These signs indicated fresh plastic exudation in the left pleural cavity, as a result of which attachments had formed with the pericardial sac, and with the lung, altering the heart sounds and giving an intra-ventricular murmur at the apex. During the rest of the month of May and of June following there was progressive plastic exudation, invading more and more of the pleura, and causing distressing dyspnœa.

From the first there had been albumen in the urine, with some casts. But in July there was notable improvement in all the symptoms, except that the râles

remained, and exercise was exhausting. In the latter part of August he returned to the city and attended to some professional duties; was out riding daily, and visited the hospitals. But late in the autumn one chilling day, at the hospital, he took cold, and was again obliged to keep his room. There was another advance in plastic exudation in the left side; the heart was more restrained by tightening bands of adhesions; there was general and gradual failure in health until the 6th of March, 1877, when he died.

*Autopsy* by Dr. Abbe.—"March 7, 1877—*Pleura:* Each cavity contained about a pint of clear serum.

"*Right lung*, bound by old plastic adhesions over pectoral and infra-mammary regions and to the pericardium; latterly over entire axillary region, and somewhat below, though not to the diaphragm. Posteriorly along spine up to the summit of the lung, where the apex was completely adherent.

"*Left lung.*—Apex adherent and firm, thence extending along the spine two-thirds downwards to base of lung; also bound at upper part of subscapular region. Three or four fine bands of recent plastic extended from pericardium to left lung. The lungs were not diseased.

"*Liver* somewhat contracted and fatty. Gall-bladder contained perhaps a dozen small concretions not larger than mustard seeds.

"*Spleen* somewhat hard and fibrous, tightly adherent to extreme of left lobe of liver by old and thick adhesions; also adherent to peritoneal wall, to the omentum, and to a little of the intestines.

"*Kidneys.*—Both somewhat contracted, the right much more than the left, weighing about three ounces; both somewhat cirrhotic and granular, and containing numerous small cysts, varying from the size of a small pea to that of a bean; both congested.

"*Heart* considerably enlarged; valves ample, but somewhat thickened (especially on the left, by atheromatus changes, fatty, etc.), beginning atheroma of the aorta, though without calcareous plates.

"*Intestines* and *bladder* normal. *Brain* not examined.

"(Signed) ROBERT ABBE, M.D."

About two years before his last illness he had an attack of erysipelas, and at that time careful examination revealed no sign of kidney disease.

When first seen on that first day of April, 1876, there were no physical signs of chest disease, except slight dullness on percussion and the loss of true respiratory murmur over a space about three inches in diameter, over the back part of the middle portion of the right lung. There were no râles nor rhonchi until the night of the fifth when subcrepitant and crepitant râles appeared exactly in the place where the loss of true respiratory murmur had first been observed. After this they were never absent, but gradually extended until they covered both lungs, becoming firmer and dryer as they grew older.

It would seem that lowered vitality had placed the capillaries of all the organs in a state of paresis and stasis, whence resulted plastic exudations—a general breaking down, in which all the vital organs were sufferers.

*Case* VIII.—(Notes and autopsy by Dr. Stedman, of House Staff.)—"M. A. S., seamstress, admitted to St. Luke's Hospital, September 29, 1877. Has been feeling ill since last spring; has had cough; lost flesh and appetite. The patient is not complaining much of her chest, but comes to be treated for intermittent. She had a chill on the morning of admission.

"Oct. 10th.—Has had a chill every other day since

admission. Examination of chest to-day shows that the right lung is free in movement in front without râles, but that there are some râles and signs of thickened pleura in the lower part of this side behind. Over the left lung there are plastic exudation râles, both in front and behind. Closer adhesions (fine dry râles) below. Soft râles are heard in the upper part, but they grow harsher downwards.

"Oct. 22d.—Patient has had no chill since the 11th. At nine o'clock this evening was seized with hæmoptysis and died from suffocation before any aid could be given.

"Oct. 24th.—*Autopsy.*—Right lung free from adhesions, except at lower part, behind. Left lung bound to the chest loosely above, more firmly below, both anteriorly and posteriorly. Lung filled with tubercles (caseous concretions), and two newly-formed cavities, one at the apex and the other at the middle of the upper lobe.

"Into this latter the hemorrhage had taken place from an eroded vessel the size of a crow-quill. The bronchial tubes and trachea were filled with blood. The pericardial and pulmonary pleura were firmly adherent."

The points of interest in this case are: 1. That the interpretation of râles as denoting an inter-pleural pathological process was correct.

2. That caseous deposits in small scattered masses may fill the lung without being detected when loose adhesions shut off sound, and especially when the true respiratory murmur is feeble or absent.

When the adhesions are firm and close, sound is more directly transmitted, and the pathological condition of the lung may be more easily diagnosticated.

3. Fatal hemorrhage, pneumorrhagia, always takes place suddenly. A softened caseous deposit opens into

a bronchus, and at the same time erodes a blood-vessel of some size, and the cavity and air-passages are immediately filled with blood, and the patient dies as by drowning.

*Case* IX.—Pietro Angelo, æt. 29, Italy, sailor, admitted to St. Luke's Hospital, May 1, 1877. Had articular rheumatism, for which he was successfully treated with salicylic acid.

June 10th.—Was examined with the expectation of discovering heart lesions, but none were found; but there were signs of a cavity under the clavicle of the left side. Dry, crackling râles were found over the left side, and in the region of the heart there were a few râles synchronous with the heart-beat.

*Diagnosis.*—Cavity in clavicular region; old adhesions over whole of lung; also adhesions between the left lung and the pericardium. Right lung free. Patient says he has had cough for some time; complains of no pain, and did not think he had any disease of the chest.

June 20th.—Patient has had high temperature for a day or two. Examination shows abundant soft râles in left side, large and small, which have supplanted the dry râles synchronous with the heart's motion.

July 1st.—Patient is losing flesh, and has cough with purulent expectoration. A creaking sound is heard in the region of the heart, synchronous with the movements of the lungs and also with that of the heart.

Aug. 1st.—Patient failing; considerable expectoration, difficult breathing, hectic, and night-sweats.

Sept. 3d.—Patient complains of severe pain in the *right* side, with increased dyspnœa. Examination showed moist, tearing râles with each respiration over *right* lung, the one hitherto healthy. On the left side, in front, a harsh leathery creak is heard, but no râles

synchronous with the heart's motion, although it is evidently restrained; behind, low down, there are numerous dry subcrepitant râles.

Sept. 14th.—Died at 5 P. M.

*Post-mortem*, Sept. 15th, seventeen hours after death. —"Right side of chest: adhesions over whole lung, attaching it to the chest-wall, but soft and easily separated by the finger. Left side: the lung is firmly adherent to the chest-wall and also to the *pericardial sac*, and could be separated from them only by dissection. There is a dry tubercular or caseous deposit in upper part of right lung, and a good-sized cavity in upper part of the left.

"(Signed) T. L. STEDMAN, M.D."

It will be seen that the soft adhesions easily detached in the right pleural cavity agree in age entirely with the appearance of moist râles of Sept. 3d. The evidence is decisive, for there was no disease of the lung nor of the bronchia to cause râle. The dry harsh râles of the left side also agree in physical conditions (firmly adherent, could only be separated by dissection) in age, with the time they had been under observation. In both sides the age of the adhesions was correctly diagnosticated by the physical signs. Another very interesting fact, and of practical importance, is brought clearly into the light, viz., that of diagnosticating adhesions between the pericardium and the lung and between the pericardium and the mediastinum, by the sign of râles synchronous with the heart's motion. These signs are not uncommon, and are additional evidence of the inter-pleural origin of all râles. Cog-wheel respiration is due to adhesions between the lung and the pericardium. If the patient takes a full inspiration, the broncho-respiratory murmur will be interrupted by each beat of the heart during the in-

spiration, and also during the time while the breath is held. The motion of the heart bringing into sudden tension the adhesions, stops the respiratory sound for an instant at each beat. If the attention is fixed upon the recurrence of these interruptions it will sometimes be possible to analyze this short rhonchus, and to distinctly recognize that it is made up of fine crepitant râles. Occasionally it is heard to the right of the sternum near the cartilage of the sixth rib, and at the diastole of the heart, simulating aortic regurgitant murmur, except that it is not heard to the left of the sternum. In this position its crepitant quality may be very manifest. The adhesions are between the pericardium covering the right auricle and the right lung. When the pericardium is attached to the mediastinum, a systolic murmur of the heart may result. So that inter-pleural signs falsely interpreted lead to incorrect diagnosis as regards diseases both of the heart and of the lungs. Many other cases are recorded which furnish equally strong proof of the correctness of the views here advocated.

The late Dr. Sprague, of Fordham, at the House of Rest for Consumptives, made about forty autopsies, in which the evidences were conclusive that the localities of râles were the sites of adhesions; that the localities of adhesions, unless so tight as to prevent all motion, were always the sites of râles. Dr. Sprague's eminent ability and painstaking assiduity render his observations of great value. I am fully persuaded that if those having opportunities will note the locality of râles for the purpose of verifying at autopsies the presence of adhesions, it will become impossible to doubt the mechanism of their inter-pleural production.

What diagnostic inter-pulmonary signs have we remaining, if all the râles and rhonchi hitherto considered

as evidence of pneumonia, bronchitis, capillary bronchitis, ædema of the lung, tuberculosis, cavities, etc., are to be interpreted as of inter-pleural origin? Need we be anxious about the consistency of Nature? May we not leave that to her, resting assured that as our knowledge is increased we will become more consistent observers, and see that she is always right? It is all-important for correct diagnosis, and in the treatment and management of disease, that the physical signs should indicate the pathological conditions. The very frequent mistake of treating bronchorrhœa for bronchitis, and ignoring the inter-pleural pathological cause, until the lung is irretrievably crippled, will be avoided. If we recognize the earliest signs of plastic exudation between the pleuræ we are enabled in all ordinary cases to promote its entire absorption. But if the favorable time is allowed to pass the exuded plastic material becomes organized, and even, if but of limited extent, may be from time to time the focus of renewed exudations, until the whole lung is bound to the chest-wall. Fibrous bands also extending through the pulmonary tissues contract, as they grow older, and finally result in the miserable condition of fibroid phthisis.

Diseases of the lungs and bronchi are manifested by by their own signs, excluding those which we have demonstrated to be inter-pleural. In so doing the gain is in greater accuracy in diagnosis, and in greater discrimination in the value of signs. The crepitant râle, although having its mechanism within the pleural cavity, is yet a valuable sign of pneumonia, or of phthisis, as it so often accompanies these diseases; but it is not pathognomonic. It may exist in the absence of both, and either may be present without crepitant râles. Centric disease, without cavities and without inter-pleural adhesions, is without râles or rhonchi,

Yet there is an area of dullness and of absence of true respiratory murmur, exactly agreeing with the locality of the disease, which, with the rational signs of temperature, pulse, and sputa, render its detection sufficiently clear to avoid mistakes in treatment. Depending upon crepitant râles as pathognomonic has many times delayed prompt treatment, and has resulted perhaps in the loss of the patient. Convincing demonstrations alone changed my views as to their intra-pulmonary mechanism. In pneumonia the exudation of plastic matter into the connective tissue of the true respiratory system is an early phenomenon. I formerly believed that the stiffened air-sacs, yielding reluctantly to the expansive force of inspiration, must separate the newly-exuded fibrine in the cellular tissue, thus giving rise to multitudinous râles. This is true only when there is a direct conduction of sound.

Dr. Walshe once proposed the same theory, which has so many plausible facts to support it, but was obliged to modify his opinion, as I have since done mine.

He found that crepitant râles, in some cases, could be proved to be due to the presence of thin fluid in the pleural cavity (Walshe on "Diseases of the Chest," pp. 107 and 108, 3d edition.)

In Dr. Chamberlain's case of atheramatous aorta (reported in the *New York Med. Journal*, Oct., 1874) I had the privilege of making a careful exploration of the patient's chest not long after the first serious symptoms were manifested. Over the lower part of the right lung there was crepitus or fine subcrepitus, and at the autospy blood was found in the right pleura, but both the pleura and lung were healthy.*

* Adhesive fluids over the moving surfaces of the pleura give rise to râles.

The signs of bronchitis of greatest diagnostic importance are not râles, but raised temperature, quickened pulse, with harsh and sibilant respiration, which masks true respiratory murmur (it does not supplant it as is done in pneumonia), with appropriate rational signs.

When resolution takes place, then true mucous râles are heard in the upper bronchi, distant from the ear, intermittent at longer or shorter intervals, as the mucus is collected or expectorated. Bronchitis may be complicated with pneumonia or pleuritis, in which case the signs will be more or less blended.

Sympathy between the bronchia and the pleura is very intimate. Severe bronchitis is apt to induce plastic exudation between the pleuræ, and plastic exudation is accompanied more or less with bronchorrhœa. Foreign bodies in the bronchia induce plastic exudation between the pleuræ even sooner than they do pneumonitis.

Capillary bronchitis may or may not be accompanied by râles; when so, the râles have their origin within the pleural cavity, and when there is no exudation there are no râles. This is a disease peculiar to children, and is really pneumonitis and has the same signs. That which is generally called capillary bronchitis, on account of the sign of small, moist râle, is simply an inter-pleural plastic exudation, to which children are also very liable.

Fine subcrepitus may or may not accompany pulmonary œdema, but only when there is exudation within the pleuræ.

The only true sign of pulmonary œdema is dullness under percussion. It is not distinguishable from pleuritic effusion except when there are fine subcrepitant râles as well, showing that the pleural surfaces are in coaptation and covered with lymph.

The diagnostic signs of inter-pleural pathological processes may be briefly stated thus: Physical signs—râles or rhonchi; large gurgling; soft, tearing, harsh, dry, rattling, crackling, small, fine, creaking. Percussion note: flat, parchment-like, wooden, high pitch, dull. Rational signs: quickened pulse, hurried respiration, dyspnœa, asthma, short, hacking cough when the adhesions are over the summit and upper part of the lung; spasmodic and strangling when in the lower pleuræ. Bronchorrhœa, hæmoptysis, irritable stomach, dyspepsia, emaciation, loss of strength, frequent perspirations, especially when sleeping; and lastly, when advanced and extensive, all the signs peculiar to fibroid phthisis.

## ARTICLE VI.

### On Hæmoptysis.

Hæmoptysis may be divided into two kinds, according to the source of the hemorrhage. It may be simply an exudation through the capillaries of the bronchial arteries of the mucous membrane—bronchorrhagia—or it may come from some eroded branch or branches of the pulmonary artery within a fresh excavation—pneumorrhagia.

These sources, though both in the respiratory system, are yet widely different in their origin, and the hemorrhages differ equally in their character, significance, and danger. One comes from the systemic circulation, the other from the pulmonic. One signifies obstruction in the capillary circulation within the lungs; the other that there is destruction of lung substance. One is not necessarily attended with danger, the other threatens instant death. Different and yet similar as are these two varieties of hemorrhage, they are both described to the popular mind rightly enough by the common term "bleeding at the lungs," a phrase which conveys to the people an idea of horror. The few cases of sudden death from hemorrhage of the lungs are published far and wide, and are ever remembered. When one spits blood, it is but natural that all interested should fear that his may prove one of the fatal cases. Public fear, as well as other forms of public opinion in regard to medical subjects, have their origin in the profession. Through the profession we hope to so instruct the common mind as to prevent unreasoning fear, and in this way it seems possible we may save many valuable lives.

Only a small number of cases of blood-spitting are fatal of themselves, and yet many of them, otherwise without danger, are the beginnings of fatal illness, because of the fright which they induce and of the wrong treatment of which they are the occasion. The anatomical explanation of these facts is the key to our comprehension of the whole subject. The bronchial mucous membrane, as well as the fibrous sheaths of the bronchi, are supplied with blood by the bronchial arteries, which blood is returned by the bronchial veins, and, so far, the analogy of this circulation to that of the body is maintained. But, in addition to this, there are in the lungs arteries arising from the bronchial, perhaps receiving supplies also from the intercostal and the mammary. These arteries go to the parenchyma of the lungs, and are the proper nutrient vessels of the true respiratory system. ("Waters on the Human Lung"—Reissiessen, Cammann, etc.)

In this place it is not necessary to show that they alone provide nutrient blood for the whole true respiratory system, which may be in part fed from the aërated blood of the capillaries and venous radicles of the pulmonary artery and vein. The exact truth on this subject may never be known. The singular fact remains that these nutrient arteries have no *venæ comites*. Their blood makes a short cut, as it were, through the capillaries of the true respiratory system, and becomes aërated even while doing its work, and is then passed immediately into the radicles of the pulmonary vein, never going through the right heart at all, and may be said never to have left the systemic circulation. This anomalous fact in the vascular anatomy of the respiratory system explains how bronchorrhagia may be caused by obstruction of the pulmonary circulation. Blood hindered or arrested in the

pulmonary capillaries obstructs the blood in the nutrient arteries. These having no veins accompanying are damned up and the blood is thrown back into the bronchial arteries; and these again relieve themselves from the accumulation by straining it through their own vascular walls, and hence this is called bronchorrhagia. This form of hæmoptysis is a safety-valve arrangement, and its great object seems to be to prevent injury to the true respiratory portion of the lungs. It not only prevents immediate injury, it does more; it stimulates the organic life of the true respiratory system, and the absorbents to take up and carry off the obstruction, whether it be plastic exudation upon the pulmonary pleura, or tubercle in the air-sacs. Bronchorrhagia may arise from two kinds of pulmonary obstruction—from that which is temporary and extrinsic to the pulmonary circulation and also from that which is more permanent and which has its seat *in* the respiratory system. The first cause, or extra pulmonic, may be from cardiac disease, hysterical passion, great emotion, or extraordinary exertion. The second is from obstruction to the circulation through the pulmonary capillaries, as by plastic exudation upon the pulmonary pleura, tubercles in the air-sacs, vesicular emphysema, cirrhosis of the lung, cancer, or benign tumor.

Plastic exudation within the pleura is a frequent and too generally an unrecognized cause. The physical signs of plastic exudation have either been called subcrepitant—sometimes mucous râles, or such as were supposed to indicate capillary bronchitis—or œdema of the lungs. I shall not here attempt any discussion of these points, as that has been partially done in a previous paper (Dr. Brown-Séquard's "Archives of Scientific and Practical Medicine," March, 1873), but shall

merely state that in bronchial hemorrhage, it is practically safer to consider these râles as always indicating plastic exudation within the pleura, notwithstanding any predetermined views that may be held upon this subject. This is not unimportant, nor is it intended to bar discussion, as will be obvious, it is hoped, when we come to speak of treatment. The rationale of this cause may be readily understood if we consider that plastic exudation upon the pulmonary pleura, in addition to the pressure caused by its presence, soon applies that cause effectually by its contraction, and so obstructs the circulation of the pulmonary capillaries immediately subtending the pulmonary pleura.

Tubercle and its inflammatory results are important causes of bronchorrhagia, the more especially as the hemorrhage may draw *early attention* to the disease, which might otherwise have remained latent until softening and disintegration of tissue had commenced. There is no controversy as to the importance of early knowledge of tuberculosis, nor as to its correct management from the beginning. Gray tubercle—Bayle's tubercle—*true tubercle*—may be so deposited as to obstruct the pulmonary circulation without furnishing physical signs, except such as are obscure to the general observer and liable to misinterpretation. The expert auscultator can alone read them with certainty. In both tubercle and plastic exudation within the pleura the capillary circulation of the true respiratory system is impeded. Hemorrhage relieves not only the immediate circulation, but it also stimulates the absorbents to remove the obstruction, whether it be tubercle or plastic lymph. From a pretty long observation of clinical facts I feel warranted in stating my conviction that I have witnessed this conservative process in not a few cases. The thoroughness of the

cure may depend upon the promptness and amount of the hemorrhage. This is especially so in first attacks.

When either of the important causes named are complicated with inflammatory products in the pleura or in the lung, the results of older disease, the removal of these products may not be so complete; yet even then a large hemorrhage may do very much to clear them away.

Vesicular emphysema gives rise to another variety of obstruction to the capillary circulation in the lungs. The true respiratory system consisting of bronchioli, infundibuli, and air-sacs becoming dilated, bronchorrhagia sometimes follows. The obstruction to the capillary circulation in this dilated state of the true respiratory system results from the elongation and narrowing of the capillaries, so that the blood flows slowly or not at all. In old cases of emphysema some of the air-sacs become destroyed, which still farther interferes with the circulation, both in the pulmonary and nutrient arteries, but with less liability to hæmoptysis, as the system, in old cases, becomes accustomed to the crippled condition, and accommodates itself to it.

Cirrhosis of the lung or fibroid phthisis is another variety of structural change, frequently accompanied by hæmoptysis. It may commence in childhood or early life, or it may be diffused, and this history assists in making a correct diagnosis. The similarity of its physical signs to those of tubercular phthisis in the third stage makes it necessary that we should treat of it somewhat particularly. In both, there may be dullness under percussion, cracked-pot sound, and cavernous respiration, when there are excavations or dilated bronchæ, which, if in the upper part of the lung, are sometimes accompanied by mucous gurgling. There will be also in both cases absence of true respiratory

murmur, altered and exaggerated broncho-respiratory murmur, and there may be bronchial or tubal breathing. There may be also these rational signs in common; hæmoptysis, cough, expectoration, and dyspnœa. They differ in history and in many of the rational signs. Hectic, loss of strength, night sweats, emaciation, and disease or disorder in other organs, liver, stomach, etc., all belong to phthisis, but not to cirrhosis. In phthisis the amount of circulating blood gradually diminishes, not so in cirrhosis; there is no cachexia in cirrhosis, but it is generally marked in phthisis. Yet the diagnosis of cirrhosis, during an attack of hæmoptysis, must be difficult. A few hints may assist in differentiation. The cavities in cirrhosis are always of moderate size and regular shape, and consequently the cavernous respiration is smoother in character and softer in quality than that formed in the irregular excavations of phthisis. The sound runs along the open bronchus each way in cirrhosis more readily, and is conveyed farther from the cavity than in phthisis. The condition of the digestive organs, fullness and vigor of the capillary circulation, but above all, the history of the long continuance of the case, enables us to make a correct diagnosis. The pathogenesis of cirrhosis is considered to be as follows: inflammation of the bronchial mucous membrane extends to the enveloping fibrous sheath, and interstitial plastic exudation takes place, which, contracting, results in stricture of the bronchus, and dilatation follows behind the stricture. In new attacks of bronchitis, plastic matter is thrown outside of the sheath, enveloping and destroying some of the air-sacs and bronchioles. As the child grows older, the physical signs give evidence of cavities and consolidated lung. This condition of the bronchi becomes more or less

complicated with plastic exudation within the pleura, which, suddenly interfering with the capillary circulation of the peripheral air-sacs, may be followed by bronchorrhagia.*

Cancer and benign tumors within the chest may also cause hæmoptysis by mechanical pressure, obstructing the pulmonary circulation. But the cases are rare, and it is not necessary to dwell upon them, for, if there be obscurity at first in the physical signs, the rapid progress of malignant disease soon renders the diagnosis clear.

*Pneumorrhagia.*—The second grand division of this subject is important on account of its great fatality. Fortunately its class is small, yet the terror which it excites is not unlike that produced by the cry of fire in a large assembly, jeopardizing many more lives by the fright and insane action which it induces than by any intrinsic danger in the thing itself. Pneumorrhagia implies an eroded branch of the pulmonary artery. For practical purposes we shall consider it merely as the effect of erosion from disease, presupposing a cavity to have been formed. Yet, even in the third stage of phthisis pneumorrhagias are rare among the number of hemorrhages. Pneumorrhagia is possible after the rapid formation of a cavity, and becomes probable if the cavity be near the root or centre of the lung, where the vessels are large and abundant. The rapid disintegration of tissue renders the branch of the pulmonary artery which passes across or through the

* There is a distinct difference between fibroid phthisis and cirrhosis of the lung. Cirrhosis has no cavities, no dilated bronches, creaks under the knife in post-mortem section, is of syphilitic origin and is a rare disease. When this paper was written, cirrhosis of the lung was not differentiated from fibroid phthisis.

decaying mass liable to morbid change, which may take the form either of disruption, resulting in sudden death, or, by shrinking up, become impervious, and so remain for years. It is then a matter of great moment to watch the formation of cavities, in what may be called a dangerous position, and to so guard the patient during the unavoidable process as to render this accident of minimum probability. Severe coughing should in such circumstances be allayed by opiates, which before perhaps had been properly avoided. Exertion and emotion should not be permitted to unduly distend the pulmonary vesesls. Small blisters may be constantly changed from place to place over and around the forming or formed excavation, in order to glue the pulmonary and costal pleura together, preventing motion in that portion of the diseased lung long enough for a clot to be firmly formed in the artery. Nature sets us the example, for no cavity appears near the surface of the lung but that its immediate vicinity is bound down by adhesions.

Reisseissen, Marshall Hall, and others, taught that the anastomosis of the pulmonary arteries was general and free. Dr. Cammann (*N. Y. Med. Journal*, 1848), in his experiments, by a series of injections into the blood-vessels of the lung of the sheep, proved clearly that this was a mistake; that instead of there being free anastomosis, there is properly none at all. Each lobulette is a complete type of the lung and has no anastomotic connection with any other part, so that, ordinarily, cavities in phthisis, gangrene, or abscess, are rarely accompanied by pneumorrhagia, and only so by the accident of their opening a branch of the pulmonary artery. If the cavity just formed is centric, with no pleuritic adhesions, the rational signs may have been so slight as not to have drawn attention. The patient may be

in attendance upon ordinary duties when the first mouthful of blood is raised. A lady-teacher in one of the public schools is standing before her class, giving instruction, when she raises a mouthful of blood, and immediately retires to a side class-room; the blood pours from her mouth and nose and she falls to the floor, and is dead before a messenger can be despatched for help. A gentleman in his bed-chamber touches the bell, and comes to the head of the stairs with a vessel in his hand, and calls to the family that he is spitting blood, and desires a physician. They hasten to his assistance, but he has fallen upon the floor and is dead when they reach him.

Sudden deaths from pulmonary hemorrhage are comparatively rare. Cases of pneumorrhagia even may not be immediately fatal; there may be several attacks before the patient succumbs. The cavity may be small, and the open branch small, or the motion in the diseased lung may be resisted by pleural adhesions. In the latter case it is possible the hemorrhage may be arrested permanently. A clot forming in and filling a cavity in a portion of lung restrained from motion may remain there long enough for the eroded artery to shrink and become impervious. But even if the cavity be small, and the arterial branch small, the frequent recurrence of hemorrhages may finally exhaust the patient. This form of hemorrhagia may be mistaken for bronchorrhagia occurring after extensive plastic exudation within the pleura. But with the absence of cavernous respiration or signs of consolidation of the lung, and with the presence of plastic or sub-crepitant râles the diagnosis need not be uncertain.

*Diagnosis.*—It needs no argument nor further accumulation of evidence to show the great value of a clear and correct diagnosis of hæmoptysis. It is of

the first importance to diagnosticate between bronchorrhagia and pneumorrhagia. In a large proportion of cases the medical attendant may state in the most emphatic manner that there is no danger, and refuse useless or harmful medication. His presence and the confidence which he inspires relieves apprehension and fright, and by wise advice he may prevent serious consequences. The chances are—perhaps a thousand to one—that a case of hæmoptysis is bronchorrhagia. It is best to assume this until a certain diagnosis proves the contrary—which turns on the presence of cavities and consolidation of pulmonary tissue. If there be no cavernous respiration and no evidence of consolidation, and, in addition, if respiratory murmurs can be heard throughout the lungs, it is clear that there can be no cavities. This determines definitely for the larger number of cases, and those not thus determined may be classed as doubtful in virtue of the same negative evidence. By farther examination we may satisfy ourselves, almost with certainty, that a large majority of these also are cases of bronchorrhagia. If the walls of the cavities are hard and unyielding it amounts almost to a demonstration that the hemorrhage is not pneumorrhagia, for the hardened walls are a sign that the cavities are old, and the branches of the pulmonary artery that traverse them are probably impervious. If cirrhosis of the lung be diagnosticated, it is certain that the hemorrhage cannot be pneumorrhagia. When a cavity is forming, or has just formed, without previous disease in that portion of the lung, if the position be central, the progress of the disease rapid, and the lung free in its motion, not held by adhesions, the danger from pneumorrhagia is imminent and should be guarded against.

*Treatment.*—Pneumorrhagia may be so speedily fatal

that there will be no time for ordinary remedial measures. It has been suggested that it might be possible to arrest hemorrhage from the lungs likely to be fatal by standing the patient upright until syncope should take place, and then placing him in a recumbent position, so that a clot might form in the cavity. I am not aware that this plan, which certainly has plausibility to recommend it, has ever been intentionally tried in practice. But we know that some of the patients were standing upright when the bleeding began, and fell from exhaustion or syncope, and so had the advantage of this hypothetical method without avail. The appalling cases of pneumorrhagia arise from central cavities of large size and from the erosion of a large vessel. Should a clot even be formed and the patient recover from syncope, the unhindered motion of the free lung would soon dislodge the clot, and the hemorrhage would again commence. Unfortunately the history of these cases shows that they are too speedily fatal for a clot to form. In a small cavity, or in a smaller open vessel, there is more likelihood of the formation of a clot, and the temporary arrest of the hemorrhage. But even here, when the cavity forms in the centric or dangerous locality, the clot is soon dislodged and the bleeding recommences, and this alternation continues till the patient sinks exhausted. In this variety of pneumorrhagia, however, there is time for something to be done; the danger is from recurrence of bleeding by displacement of the clot. If this can be prevented long enough the vessel may become impervious and immediate death be avoided. There can be no hope to fill the indication but by mechanical interference; medicines have no power over open vessels in the centre of the lung. Our experience suggests that nature has here pointed the

direction that our endeavor must take to afford any hope of success. Motionless lung alone can permit the closing of the vessel. Nature secures this condition by adhesions—we can in some degree imitate her by transfixing the lung in the diseased part, thus holding it still till the danger is past. Needles of platina or gold, passed from different points through the lung, may successfully prevent motion, and they might remain, doing no serious injury, until inflammatory action should be set up, and thus effectually prevent a recurrence of hemorrhage. Or the needle of the aspirator could be passed into the cavity and an injection of a drop or two of liquor ferri persulphatis or other powerful styptic could be thrown in, which would form a firm clot in the open vessel as well as in the cavity, and the hemorrhage be permanently arrested. This presupposes an accurate diagnosis of the *position* of the cavity. As before said, fatal pneumorrhagia is not likely to occur near the surface of the lung, where the contiguous pleura is bound down by abundant adhesions, and it is a wise precaution to frequently apply small blisters over and around forming or recently formed cavities. This measure not only guards against pneumorrhagia, but it prevents a cavity discharging its contents into the pleura, causing hydro-pneumo-thorax. Effusion of serum into the pleural cavity arrests pneumorrhagia by mechanical compression, in the same way as we sometimes see it crush out pneumonia. This condition might be artificially produced by pumping water into the pleura. Ligaturing the limbs with tourniquet or strap is easily performed, and it is a practical and frequently used method of arresting hemorrhages, for it prevents the return of venous blood to the right side of the heart, and temporarily relieves the pressure on the pulmonary circulation. The hemorrhagic act is

preceded and accompanied by an eager hastening pulse-beat, which the medical observer soon recognizes, and advantage may be taken of this monition to gain time for the application of mechanical means. If some of these means suggested seem to be harsh, farther consideration will show that they are not so in reality, and it must be remembered that the occasion is desperate and none other are of any use or promise any hope. Medicines given by the mouth, or otherwise, can have no control over a bleeding artery in the centre of the lung.

In bronchorrhagia there is no danger of sudden death, and consequently there is time to select the best methods of treatment. When the cause is extrinsic to the lungs the remedy should be to the cause and not to the symptoms. In hysteria the treatment should be to the disease, and the same in cardiac affections. The hemorrhage *per se* gives relief, and when left to itself it may be safely said is never other than beneficial. After an hysterical hemorrhage from the lungs, which may have produced alarming fright in the bystanders, the patient may fall into a gentle and placid sleep, for which she awakes relieved.

Bronchorrhagia arising from obstruction to the pulmonary circulation from a diseased heart must be treated by paying attention to the heart alone, endeavoring to give it force and power to equalize the circulation. Stimulating enemata, brandy by the mouth, mustard foot-baths, the sedative action of calomel, digitalis and nux vomica, may relieve the heart from its oppressed condition, and the hemorrhage will cease naturally. Bronchorrhagia resulting from obstructed pulmonary circulation, caused by plastic exudation upon the pulmonary pleura, is of more frequent occurrence than any other, and its mismanagement may be

followed by the most serious consequences. The lung oppressed by the presence of plastic exudation is more and more crippled by contraction. A large hemorrhage at the beginning is frequently followed by the immediate removal of the cause by stimulating rapid reabsorption. Attempts to arrest this hemorrhage are generally futile, but the evil results of the methods used and the delay in applying proper remedial measures may end in what is popularly known as "hasty consumption." The old custom, lately reinstated by the translation of a popular German text-book,* is to place the patient in bed in a semi-recumbent position, to direct that he be kept quiet, to speak only in whispers, not to cough, to darken the room, and prevent all motion that may be avoided. In addition to this medicines are given *to stop* the hemorrhage, as acetate of lead, opium, kino, tannic acid, gallic acid, mattico, ergot, spirits of turpentine, and table-salt. In spite of all this treatment and mismanagement the bleeding continues more or less at intervals, causing prostrating fear to the patient and agony to the loving attendants. This may go on for days or weeks, until, nature giving up the contest, the bleeding may cease, only to be followed by disintegration of tissue, and death. An entirely opposite treatment might have saved life. But instead, the effort of nature is thwarted and rendered of no effect. The threatening conditions, which she would have changed if she had not been prevented, are carefully preserved, and in addition to all this the exuded blood is kept in the bronchi decaying and offensive, causing local inflammation and peribronchial abscesses. Medicines which do harm to the patient, and do not arrest the bleeding, are resorted

* Niemeyer.

to, while all that should be done is left undone. It is no wonder, viewed in this light, that "hemorrhage causes phthisis." Would it not be better to say, rather that the physician cultivates phthisis? As illustrating the possible consequences of repressing all effort to relieve the clot in cases of hemorrhage, there is an instructive case in Graves, who reports the incident of a gentleman who had several hemorrhages in one day, and was visited by Dr. Stokes, who found him collapsed (he had also been bled from the arm) and well-nigh asphyxiated; the right side of his chest was expanding and contracting energetically, the left almost fixed and motionless. Dr. Stokes changed his position and gave him a glass of wine, when he made an effort and violently expectorated a fibrinous coagulum forming a complete mould of the left bronchus and its ramifications. Loevenhard, quoted by Valleix, cites a case of a woman to whom alum had been administered in hæmoptysis with the hope of arresting the hemorrhage, and apparently with effect, but upon the cessation of the bleeding, suffocation became imminent; and from this danger the woman was only relieved by the rejection of a large quantity of coagulated blood.

What, then, should the physician do when called to a case of this nature? His first determination should be *to do no harm*. He should, by his cheerful, confident demeanor, inspire the patient and friends with his own courage. Then, after auscultation and the diagnosis of bronchorrhagia, he should insist upon the patient's speaking aloud, that he should breathe freely, sit up, walk about, cough and expectorate freely. There is no danger of increasing the hemorrhage by coughing, loud speaking, and full respiration; these acts merely help to loosen and dislodge the blood already exuded into the bronchia, and which is there obstructing respi-

ration. It should be the object of the treatment to expel all the exuded blood before any part of it becomes putrid. Clearing out the bronchi and full respiration do not increase the exudation; on the contrary, these healthful acts go far to equalize the circulation and help to arrest farther hemorrhage. Salt and spirits of turpentine are innocuous, but do not let the patient believe that they are given for the purpose of arresting hemorrhage; it is best to stand firmly upon the ground that the hemorrhage is beneficial and should not be arrested. On this account we should refuse any medication, for, if the bleeding does not soon cease, the patient begins to lose confidence in the doctor's ability to arrest it, which may be calamitous. The confidence inspired by the presence of one believed to have power to help us, gives tranquillity of mind and that steady nerve action which, operating directly on the heart, equalizes the circulation; the cold extremities become warm, the oppressed breathing becomes free, and these facts prove that the pulmonary congestion or stasis is relieved. Many remedies have gained reputation for power they did not possess, *by the real power* of a trusted physician's presence, acting through the emotions upon the organic life of the body. As soon as a diagnosis can be made between plastic exudation and early tuberculosis, a line of treatment should be adopted not "to cure the bleeding," but to remove the cause. In both plastic exudation and early tuberculosis all the special treatment should be the same. The lungs should be frequently and systematically expanded; as much fresh air should be inspired as the system will accept. The diet should be nutritious, not overstimulating, but such as is known to be most beneficial in tubercular phthisis. If plastic exudation should be diagnosticated by finding persistent, subcrepitant or mu-

cous râles, soft-tearing and near the ear, without dullness of the percussion note, its early removal would be an object of the highest importance. Nothing helps us in this object more speedily, perhaps, than the bleeding itself, and for this reason we should not arrest it, even were it within our power to do so. The beneficent and harmless action of calomel when used for its sedative effect, in cases of commencing or threatened inflammation, is not so well known as it deserves to be. In "Graves' Clinical Medicine" (pages 803-806 inclusive, ed. 1843, Dublin), its qualities and capabilities are well set forth. In Dr. Lente's paper (*N. Y. Jour. of Med.*, May, 1869), its successful and safe application in dysentery and some other inflammations is ably maintained. In recent plastic exudation, followed by hemorrhage, its beneficent and prompt effects are more remarkable than they are in any other inflammation. It wipes out, as it were, all evidence of disease, the hemorrhage ceases, the subcrepitant and mucous râles disappear in an incredibly short time, leaving the patient well. It has been unfashionable of late years to speak of calomel and bleeding as proper remedies in any case. No doubt they have been abused, and so too have many other good instruments which have not been discarded.

Above all other things it is important that specifics or styptics, of whatever name or character, especially those that may depress the vital power or derange the digestive organs, should be *withheld* in bronchorrhagia. Our one object should be to remove the cause.

NOTE.—The foregoing article was read before the *New York Journal* Association, 1875.

## ARTICLE VII.

### Endemic Pleuro-Pneumonia, as seen in New York during the Past Ten or Twelve Years.*

Pneumonitis, such as may occur from a wound of the lungs in a person in previous health, even when followed by grave constitutional disturbance, is essentially different from the typical disease called pleuro-pneumonia, which is but the exponent of a series of systemic perturbations and changes resulting from differing and mixed causes.

Endemic pleuro-pneumonia in New York during the last ten or twelve years has had distinct and peculiar factors, some of which were known or partly understood while others were unrecognized or obscure.

We are apt to fix our minds upon the obvious and immediate, thus perhaps directing our attention away from the hidden causes which may give disease its peculiar characteristics.

Cases of ordinary pneumonia may have varying elements. That with malarial tendencies differs from that with typhoidal, and where these are combined the resulting disease has characteristics essentially its own.

In the epidemic ship fever in New York in 1845 to 1854 the complications by pneumonia were not rare, nor were they exceptionally fatal in their tendencies unless influenced by peculiarities of nationality. The German poor were not so liable to the fever as the poor Irish, but in them pleuro-pneumonia was a much more fatal complication.

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* Reproduced from the transactions of the Medical Society of the State of New York for 1880.

For many years a form of fatal pleuro-pneumonia has prevailed in the cities of the Southern States, while in the North there was another form of this disease of mild type and easy management. At the same time there was a gradual procession of the fatal form northward overrunning Washington, Baltimore, and Philadelphia, until, about 1868, it reached New York. A comparison of statistics will show to what extent it has influenced the bills of mortality since then.

In 1839, in a population of 301,697, the whole number of deaths was 7,361; from pneumonia and bronchitis, 568. I have included bronchitis, as I have no doubt that the cases so reported were mostly if not all pleuro-bronchial or pleuro-pneumonic. In 1851 there was a sudden and noticeable increase of deaths from these diseases. In a population of 545,359 the whole number of deaths was 20,738, and from pneumonia and bronchitis 1,569.

In 1856, in a population of 694,607, the whole number of deaths, was 20,102; from pneumonia and bronchitis, 1,159, which was a falling off.

In 1868 there was again an increase in the death rate from pneumonia and bronchitis. In a population of 913,298, the whole number of deaths was 24,889 and from pneumonia and bronchitis, 2,471.

In 1875, in a population of 1,041,886, the whole number of deaths was 30,709, and from pneumonia and bronchitis, 3,913.

In 1876, population 1,055,535, whole number of deaths was 29,152; from pneumonia and bronchitis, 3,756.

In 1877, population 1,069,362, whole number of deaths 26,203; from pneumonia and bronchitis, 3,181.

In 1878 population 1,083,371, whole number of deaths

27,008; from pneumonia and bronchitis, 3,472. The fatality being greatest in 1875.*

There is and has been since the endemic appeared a dread of it in the public mind of New York hardly exceeded by that of any of the great epidemics which have prevailed heretofore.

The profession too, at its commencement, had reason to be alarmed, for many of the cases ran so rapidly toward a fatal termination that curative measures were useless before the gravity of the case was comprehended. The disciples of Hahnemann, as well as those who depended entirely upon the "vis medicatrix naturæ," were astonished at the powerlessness of their feeble efforts.

In connection with this subject, and perhaps as explanatory, we may go back to the history of an epidemic of pleuro-pneumonia which had its origin in Canada during the war of 1812-15.

It first attracted attention in the British army by its contagious element and great mortality. It soon invaded the American lines and decimated the raw troops unused to camp life. Many of the sick (hospital accommodations being poor) were given leave of absence and carried the contagion to the cities and even into sparsely settled country districts.

It followed the course of travel southward, reaching Charleston in 1818 and the cities of the Gulf in 1820. Here it lost its contagious element, ceasing to be epidemic, and becoming endemic remained as one of the specially fatal local diseases.

Dr. Samuel Henry Dickson has described it graphically as "pneumonia typhoides," which in its active

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* Compiled from the records of the City Inspector by John T. Nagle, M.D., Dep. Registrar of Records.

state must have been similar to the "pleuro-pneumonia contagiosa" of the cattle herds of to-day. It was known among the people as "the epidemic," and those who recovered from it were subject to "bronchial affections" ever afterwards.

From these and other facts, and with our present knowledge, we may assume that the disease, like the cattle disease, was largely characterized by extensive exudation of plastic material into the pleural cavities.

It may not be possible to prove that endemic pleuro-pneumonia, as we know it in New York, is the legitimate successor of the contagious pneumonia-typhoides of 1812-15, but there certainly is more than a probable connection, and in seeking for the effective hidden causes of the mortality of this disease we cannot dismiss the careful consideration of its inherited tendencies as well as of those which are known to be temporary and the immediate consequences of unusual but varying conditions. As illustrative of the necessity of considering all relative facts, we may cite the example of the celebrated Dr. John Huxham, of Kent, England. He observed the weather and kept careful records of the wind, temperature and rain-fall in order to connect these meteorological conditions with the prevalent diseases, especially with those which were epidemic.

The first volume recorded observations from 1727 to 1737, and the second from 1738 to 1747 inclusive. Afterwards he published essays on fevers, small-pox, pleuro-pneumonia, pleurisies and "ulcerous sore throats." These grew out of deductions consequent upon making his weather observations and noting the behavior of diseases under these influences. The first chapter of his dissertation on "pleurisies and peripneumonias" is of the power of the winds and seasons in producing these distempers.

He quotes Hippocrates—"cold, northeasterly winds bring on disorders of the breast, sides, and lungs," and says: "This hath been found constant and true by all his successors."

"Not but that pleurisies and peri-pneumonias especially are frequently observed in other constitutions of the air, the latter very often supervening on other acute fevers. Yet still it is certain these two diseases are much more frequent when a cold, dry season and northerly and easterly winds have continued for any considerable time." He also says: "It is a fact," cæteris paribus, "blood drawn in such prevailing constitutions of the atmosphere is constantly found more dense and viscid than in long, moist, warm seasons." In his history of the epidemic of "ulcers in the throat," he records the state of the weather for many months before the advent of the disease. "The weather was pretty cold and dry in March, 1752, especially at the beginning and latter end, and the barometer high. At no time very low.

The small-pox grew more mild and much less frequent. The other diseases also less common, but more inflammatory; no malignant sore throats; many were severely tormented with coughs and obstinate asthmatic disorders. The blood now drawn was commonly more dense and viscid than it had been for many months. At the last half of the year 1752, in summing up, he says: "For many months past we had scarce the slightest fever, but it was attended with a sore throat, aphthæ and some kind of cuticular eruption. The blood drawn from the diseased during all this time has been very rarely viscid, but generally florid, seemingly, especially at the very beginning of the malady, and of a very loose texture."

We see that Dr. Huxham was a devoted student of

nature, and his views are all the more valuable, inasmuch as he was not a slavish adherent of any school. His accurate description of "The Ulcerous Sore Throat Epidemic" of that time is true now of diphtheria, and it undoubtedly was the same disease.

Brettonneau's description is hardly better, and it is singular that he should say "Huxham's ulcerous sore throat did not pertain to diphtheria, but rather to some form of scarlatina."

It must be that Brettonneau received his knowledge of Huxham at second-hand. He could not have said so if he had read him attentively.

If meteorological records had been kept for the last hundred years, and their relation to epidemic forms of diseases noted, it is possible that we would have had a more accurate knowledge of devastating epidemic diseases and also of the best methods for their prevention and cure.

The Weather Bureau at Washington might combine with its meteoric records from all parts of the continent one of the sanitary conditions—the kind and form of epidemics and their peculiar characteristics, should any exist, and their direction and radi of progress.

Public health associations might assiduously collect such facts, which in time would determine the existence of sanitary laws of which we are at present ignorant.

Lord Bacon says, in substance, that it is the opinion of some that the conditions of weather repeat themselves in a cycle of about twenty-five years, and that there is reason for such belief. The popular opinion that periodical influences of the heavenly bodies control health has some foundations in fact. We know that extraordinary vicissitudes of weather, violent variations of temperature, winds, moisture, electricity

and malaria—and other imponderable agents influence health.

There are disturbing elements which determine the particular characteristics of epidemic diseases. Theories are abundant, but no theories are of great utility which are not confirmed at all points by facts. The germ theory supposes that the unknown factors are floating in the air, and produce disease by entering the circulation of the blood. At all events this is plausible, and may aid in directing attention to the controlling hidden cause. Dr. Huxham's method of studying the conditions of the blood, as to viscidity and coagulability, is worthy of attention, and suggests a still more accurate way by means of the microscope and by chemical analysis of prognosticating coming epidemics. Such knowledge might give us the power of meeting them at the commencement and of rendering them innocuous. Yet all does not depend upon vicissitudes of weather.

Before the appearance of Europeans upon this continent, it is said, the red men were not subject to devastating epidemics, and yet the meteoric conditions, we may infer, were not greatly different from what they have been since, except as modified by cutting down the forests and the tillage of the ground.

The vices of civilization, the aggregation of people in cities, towns and camps, are elements from which epidemics are bred. Human filth and human excesses shorten the average life of the race and increase the tendencies to disease in the individual.

In order to show the change of type in pneumonia, I will premise that when I began my professional life I believed as I had been taught, that active interference was necessary in every stage of the disease, to prevent destructive inflammation But I soon found

that cases would get well without active treatment—that the expectant plan or wise management was best—simple medication with stimulants and supporting alimentation. The mortality was so small that it seemed unnecessary for an uncomplicated case to reach a fatal result. It was the belief of eminent practitioners at that time, whose friendship I enjoyed, that the Asiatic cholera of '32 and '49, and the ship fever of '47, had modified the type of inflammatory diseases, and made the change in treatment necessary, which was then becoming fashionable. Homœopathy had an immense advantage in this change of type. It shook the faith also of some high in the profession. Sir John Forbes, of London, and Dr. Bigelow, of Boston, began to teach the doctrine of self-limitation of diseases and the inutility of active medication, and gained many adherents. When the fatal forms of pneumonia began to occur in New York, about ten years ago, I, with other physicians, was surprised at the failure of the managing method, and the frequency of fatal terminations, and I became impressed with the truth of the proposition that with the change of type there had been also changes in the pathological process. The cases were more "typhoid," or of depressed vitality, and the inter-pleural complications more frequent and of graver import. Indeed the increase of mortality was measured by these complications. Some method of treatment more efficient was necessary.

The late Dr. George P. Cammann, whose eminently practical mind led him to investigate the peculiarities of each case by itself, had taught that in cases of great and sudden congestion of the lungs—the very conditions which we now recognize as indications of inter-pleural complication and rapid plastic exudation—very large doses of calomel, used promptly, would control

and prevent a fatal termination. After his death I caused to be printed a paper of his, which he denominated "Sanguineous Congestion of the Lungs," in which he related cases treated by this heroic remedy. The dose used would vary from ten to sixty grains, according to the urgency of the case. Dr. Cammann's cases were accidental and occasional, but no doubt were the same as the endemic pleuro-pneumonia of to-day. The inefficiency of the expectant method rendered a resort to the heroic a necessity, and with very encouraging results. Cases, such as had proved fatal by the mild treatment, were saved by the prompt exhibition of sedative doses of calomel, which are less depressing to the system than smaller doses repeated. I am aware that Mialhi has stated that more than ten grains of calomel is a waste of good medicine. This statement has been repeated by Headland, and is constantly quoted by those who oppose the use of calomel, having no practical knowledge of its wonderful efficacy more than the authors quoted, who evidently knew nothing at all. But the difference of life or death many times depends upon the prompt exhibition of doses many times larger than ten grains. With the immense prejudice operating against the use of calomel, it requires the courage of experience to give the very large doses—twenty, thirty, forty, or even sixty grains. The medicine is not thrown away, and it is safer to give a few grains more than might barely do than to repeat the dose.

In Graves's "Clinical Medicine," Dublin, 1848, p. 803, he says: "If a person is seized, for example, with very acute pericarditis, how unavailing will be our best directed efforts unless they be seconded by a speedy mercurialization of the system. In proof of this assertion I might adduce a considerable number of cases of

pericarditis, treated both in hospital and private practice, and might triumphantly compare the results with those obtained in the Continental hospitals, as recorded by some of the most eminent German and French physicians."

Dr. Graves discusses the arguments of those opposed to the free use of mercury, who acknowledge it may be necessary to use it in hot climates, as recommended by Dr. Johnson in his classical work, but who deny its utility to Europeans, but he answers that, "this observation no doubt deserves attention; but its weight falls to the ground if experience contrary to the generally received opinion shows that, with proper precautions, calomel may be given in as large doses here as in the East Indies." And again he says, "another most important question is, whether mercury so used for the cure of internal inflammations injures the constitution permanently. With the greatest confidence I can answer it does not." I never saw a single bad effect follow the use of mercury, in cases where the first consequences of its exhibition was the rapid and complete removal of a dangerous inflammation." To all of these strong expressions of one of the greatest clinical teachers the world has ever known I can give my entire assent as the uniform result of my own experience. Dr. Graves would not use mercury in either large or small doses, except from necessity, when death and permanent injury might be avoided and where no other means would be successful. It is the great remedy which may be held in reserve when all others fail. The method of giving it is dry upon the tongue. Dr. Graves advises to wash it down with thin gruel, but I believe it is best to put it on the tongue and there leave it. Its rapid effect shows that it is influential before entering into the circulation, for many times it

has a sensible effect in immediately controlling the heart's action and in reducing the temperature, while its full beneficial effects may not be had until twenty-four hours have passed. Many times the patient goes quietly to sleep who had previously been restless and sleepless, resisting anodynes. One other result of the large doses of calomel is also noted by Dr. Graves, which is, that when internal inflammation is rapidly overcome, temperature and pulse falling, with subsidence of all the alarming conditions, we may confidently expect the reparative process to continue until all is cleared up and not a vestige of the disease remains. But that this much-desired object may be obtained, the full sedative dose must be given; just enough to relieve the urgent symptoms may fail to clear up all the results of inflammation.

The safety of the large dose, in any case where it is indicated at all, is absolute; consequently the physician arriving at the conclusion that the sedative dose must be given should not fail in courage to complete the work, so necessary to be done, by any half way measures.

The following case is given in detail as typical of endemic pleuro-pneumonia occurring in New York and vicinity since 1869, and uncomplicated except by malaria and sewer gas poison:

J. R. L., physician, 58 years old, in good health; November 16th, 1878, at 11 o'clock, had a prolonged chill; about 1 P.M. he was examined by Dr. Hudson, who found crepitant râles in the posterior lower half of the left lung, with dull pain; temperature, 105½°; pulse, 160; respirations about forty. Thirty grains of calomel were placed upon his tongue at once. Temperature immediately commenced to fall—104, 103, 102, until at 7 o'clock it was 99°. However, before 10 P.M. he had

another slight chill, after which his temperature went up again to 104½°.

November 17th, before noon, had another chill, and temperature went up to 105,° with advance of physical signs. Again thirty grains of calomel were placed upon his tongue, and again the temperature began to fall. At 3 P. M. it was 104°; at 5.15, 103°; at 8.25, 102°. Monday morning, November 18th, it was 101¼°. During the day the temperature again went up to 104°, with slight advance of the râles on the right side. After that the temperature remained not higher than 101°, until the seventh day, when perfect defervescence took place.

There were no uncomfortable conditions caused by the calomel—on the contrary the relief was almost immediate; within half an hour the patient was sensible of it.

It is certain that the large doses were harmless. Possibly it would have been better had the full drachm been given at once. At all events, not only was the disease reduced to a mild character, but no adhesions remained—no disability. The entire disappearance of all signs of fever on the seventh day may be adduced as evidence of its natural subsidence.

But would it have been so had the calomel not have been used? I think not, and for these reasons. In the first place, the attending and consulting physicians, Drs. Hudson, Otis, and W. W. Jones, believed that he could not have lived until the seventh day had the calomel not have been given; and secondly, experience shows that where there is a large amount of plastic exudation, defervescence does not take place on the seventh day, but the fever continues indefinitely.

The rule of defervescence applies only to cases in which there is but little plastic exudation within the

pleura. In the twenty-three cases of pneumonia reported by the Secretary of the Committee on Therapeutics of the Therapeutical Society of New York, treated with large doses of calomel, there were twenty recoveries and three deaths.

Notwithstanding the fact that endemic pleuro-pneumonia is fatal in its tendencies, other cases occur at the same time which are of the mild type and in which the tendency is to get well, and in such mild treatment should only be used.

How are we to distinguish the mild from the more serious forms of the disease? First, by the rational signs or symptoms; second, by the physical signs. In the mild form the respiration is not greatly oppressed, and although the febrile conditions, pulse, temperature, etc., may denote much activity, yet from the fifth to the ninth day there is sudden and generally complete defervescence with or without treatment, and the convalescence is uninterrupted. While in the most serious form, there is dyspnœa from the beginning, lividity of the countenance, restlessness, and seeking the upright position. The pulse is frequent and feeble, the skin cool and moist. Temperature may run very high or may be moderate. There is no natural period of defervescence. Many times the patient dies before the fifth day.

The differences in the physical signs are equally marked. In the mild form the pneumonic conditions, sputa, etc., are sometimes well pronounced for days before the physical signs of crepitant râle and bronchial breathing appear. The crepitant râle is distinct and is not mingled with subcrepitant or larger râle, until the *râle redux* commences. In the severe variety the râles are generally mixed and begin with the disease, and the movement of the lungs is notably re-

strained. Sometimes there is but moderate dullness, but always marked flatness under percussion. The râles, too, are abundant and varied in character. All these differences of physical signs depend upon the inter-pleural complications. In the fatal variety there is a large amount of plastic exudation, generally in both pleuræ and frequently covering a greater part of both lungs. In one the pneumonitis is the principal lesion, in the other, the inter-pleural plastic exudation. Heretofore I have endeavored to show that there has been a misconception of the significance of râles as a physical sign. There are great differences in the progress of cases of the mild and of the fatal in their tendencies. The mild run an even course, and their day of convalesence can be prognosticated, and they need but little medicine.

The others have no regular course except their tendency is to a fatal termination. The hyperplasia of the blood is their distinguishing characteristic. Exudation of plastic matter into the natural cavities through serous membrane may take place, or perhaps bring the patient's life to a hurried end by the formation of a heart clot. The feebleness of the heart's action, and quickening pulse, the dusky ashen hue, cold, clammy skin and spasmodic respiration, show that death is commencing at the heart.

I have frequently demonstrated to my own satisfaction the immediate connection between these signs and symptoms of disease and the plastic pathology of the blood and its exudation into serous cavities and formation into clots in the heart and great blood vessels. But others have *not* been so completely convinced as to the direct interpretation of the physical signs as applied to inter-pleural processes, on account, perhaps, of

the time elapsing after the diagnosis had been made until its verification after death.

But during the month of August last an opportunity was afforded me of obtaining proof which the most sceptical must acknowledge to be convincing.

A commission appointed by the U. S. Government, of which Gen. Patrick is president, Prof. Lawe, of Cornell University, is a member, and J. D. Hopkins, veterinary surgeon, is inspector, has for its object stamping out contagious pleuro-pneumonia among cattle. By the kindness of Dr. Hopkins and the commission I was invited to be present at the destruction of cows, condemned by the commission, in order to examine them before death by auscultation and percussion, and to make a diagnosis to be immediately tested by post-mortem examination.

On the 19th of August, 1879, there were four cows condemned and to be destroyed on the dock, foot of 38th Street and Hudson River, New York. In each of the four cows suffering with acute disease of a few days standing, there was dullness over one lung with raised pitch in percussion. In auscultation there were râles over the affected side agreeing with the locality of pathological conditions of the pleura. Where there were râles there were always adhesions. Where the râles were dry and harsh in character the adhesions underneath were organized, and more or less firm. If the râles were moist, the adhesions were moist and cellular. Where the râles were coarse the adhesions allowed considerable movement of the lung within the chest wall; when the râles were firm there was but little motion, the lung being confined by close, firm adhesions to the chest wall. In every case the lung was completely solidified, every air sac distended with exudative matter, so that the whole lung occupied its

cavity in the chest to distension, and, when removed, was a solid cone-shaped mass, standing firm of itself and keeping its form. False membrane covered the pleura, and extended into and occupied the interlobular spaces, and the cellular tissue around the bronchæ, diminishing their calibre. Wherever there was cellular or connective tissue there was plastic exudation, and, if not very recent, was already organized, so that not only the whole lung was thus encysted, but each lobulette, a pathological condition peculiar to the bovine animal where there is so large an amount of connective tissue in the lung.

These pathological conditions of course preclude the entrance of air into the lung or its farther distension in attempted respiration, and are irresistible evidence that the râles heard were not intra-pulmonary or intra-bronchial, and therefore must be intra-pleural, their only source.

On the 27th of August, 1879, a chronic case of four months' standing was slaughtered for beef at the corner of First Avenue and Forty-fifth Street. The cow had been giving thirteen or fourteen quarts of milk per day. I was a few minutes too late to examine her, as she had been killed when I arrived, and the post-mortem examination had been commenced. The affected lung was completely disorganized by cheesy tubercular (so called) degeneration, and broken down into a pulpy mass of a yellowish, whitish color. It is termed "encysted," as the abscesses are confined by the false membrane over the pleura and in the intra-lobular and peri-bronchial spaces.

Dr. Hopkins, who is an expert auscultator, told me that the physical signs were dullness over the diseased lung, with only a few râles and rubbing sounds over

the diaphragm and over the shoulder, over sites of inter-pleural adhesions.

On the 28th of August, at foot of Thirty-eighth Street and Hudson River, on the dock, two cases were examined. One of these cases was acute, and as usual one lung only affected. There was dullness under percussion, and râles over the affected side, agreeing in locality and characteristics with the inter-pleural pathology. The lung was impermeable to air. The second case was of chronic disease of the lung, but was not known to have been of contagious pleuro-pneumonia, and was of about six months' standing. There was marked dullness over the diseased lung, and there were râles over the shoulder and over the diaphragm, but none between these points. Post-mortem examination showed adhesions where the râles were heard, but none over the middle of the lung. The whole lung was carnified and covered with false membrane. On the 30th of August one cow was examined and killed at the foot of Thirty-eighth Street and Hudson River. It was an acute case; the cow had been ill but a few days. There was complete dullness over the right lung, with bronchial breathing over the middle portion, without râles. There were râles over the shoulder and over the diaphragm. Post-mortem examination showed consolidated lung, except a portion of the under and lower part, which was œdematous—false membrane covered the lung and extended into the intra-lobular and peri-bronchial spaces. There were adhesions at the diaphragm and under the shoulder, but none intermediate, but there was an accumulation of fluid separating the pleuræ in the middle of the lung. Over the ædematous lower portion there were no râles, nor over the middle portion where the fluid separated the pleura. On the left side auscultation showed the respiratory

murmur muffled, a little roughened, but by close attention moist, almost inaudible, soft, râles could be distinguished.* Post-mortem examination showed commencing plastic exudation like thin fluid glue, moistening the pleuræ, and in some places filaments of false membrane could be raised upon the point of the knife and separated from the pleura, showing that at so early a date had organization commenced. In all these cases of the cows the proof was complete. The râles always indicated adhesions, and when there were no adhesions there were no râles.

Accepting, then, the evidence of râles, as proof of inter-pleural plastic exudation we are enabled to treat these cases commensurate with their gravity and at the initial stage, when success is best attainable.

The physical signs of râles must be searched for with earnest attention, in any case where they are suspected, and when they are discovered, the side in which they are should be supported and restrained from movement by adhesive plaster extending from the spine around to the sternum. The porous plaster is best, as it is elastic, and allows auscultation and the application of spirits of turpentine, should it be deemed necessary. Then should be considered and determined the weighty question, what is the best course to be followed in treatment. If prompt and energetic measures are decided upon, no time should be lost in putting them in force, that heavy blows may be at the beginning, not to be repeated, when the patient's strength is well-nigh exhausted. Everything afterwards should be support and building up, and by assimilable food of which milk is the type.

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* The diagnosis was hyperæmic lung with commencing inter-pleural plastic exudation.

## ARTICLE VIII.

### A New Classification of Phthisis Pulmonalis, with Reference to Special Treatment.*

The tendency of the present time is to re-arrange and to classify specific divisions of medical subjects in order to their thorough elucidation.

Not more than a quarter of a century ago Dr. Samuel Henry Dickson, one of the most accomplished and scholarly physicians of his age, and representing the advanced thought of his time, described typhus fever as one disease, with lesions of the head, with lesions of the chest, and with lesions of the abdomen.

These divisions included those which we now call typhus fever, typhoid fever, and typhoid pneumonia. But it was an intelligent attempt to bring order out of chaos.

Dr. Murchison and others have shown since, that these divisions comprise separate unities widely differing in causation, history, physical signs, and in pathological changes, and with the happy result of indicating more rational and far more successful methods of treatment.

That which has been done for the family of typhoid diseases remains yet to be perfected for phthisis.

Sydenham says: "There are several kinds of consumption. The first mostly arises from taking cold in winter; abundance of persons being seized with a cough upon the coming in of cold weather, a little before the

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* Archives of Medicine, June, 1879.

winter solstice, which happening to such as have naturally weak lungs, those parts must needs be still more weakened by frequent fits of coughing, and become so diseased at length hereby as to be utterly unable to assimilate their proper nourishment.

"Hence, a copious crude phlegm is collected. The lungs, being hereby supplied with purulent matter, taint the whole mass of blood therewith, whence arises a putrid fever, the fit whereof comes towards evening and goes off towards morning, with profuse and debilitating sweats. And when the lungs lose their natural tone, tubercles ordinarily breed therein. . . . When this disease is confirmed, it for the most part proves incurable." Is not this a good description of consumption for one two hundred years old?

Laennec and his followers classed everything in pulmonary phthisis as tubercular. "This," he says, "I think is the only kind of phthisis which we should admit, unless, indeed, it were the phthisis nervosa and the chronic catarrh simulating tuberculous phthisis."

Broussais held with the ancients that phthisis may result from inflammation, but Laennec charged him with doing so by assertion and ratiocination, however, rather than by facts. The tide of Laennec's well-earned fame has floated some errors down to our own time, especially this one which throws contempt upon the observations of his eminent compeer.

Sir James Clark, Sir John Forbes, and other English writers who had learned immediately from Laennec and Louis, followed strictly in the line of the great French leaders, and created, so to speak, *a tubercular public opinion*. But now a wider and more catholic view is being taken by English and American physicians, who are conservative and practical rather than hypothetical.

Dr. Andrew Clark, of London, in a lecture at Bellevue Hospital last autumn, and which was reported in the New York *Medical Record*, divided phthisis into three, as he said, natural classes, viz., Tubercular, Catarrhal Pneumonia, and Fibroid. Catarrhal pneumonia and fibroid are really stages of the same disease. It is best to include all forms under two general divisions, tubercular and fibroid, which represent the great natural divisions, and are descriptive of great pathological conditions and differences. In one there results death of tissues, in the other functional incapacity. In both there are cough, expectoration, and wasting—and there may be hæmoptysis, but even in these particulars, common to both, they are individually different as they are also in their grander distinctions.

Indeed, they are opposing diseases of the same organ, which, did they not frequently coalesce, producing new diseases by such combinations, would always be described as distinct.

Niemeyer, leading the modern school of pathological medicine, includes all non-tubercular forms of phthisis under the term catarrhal pneumonia, which name, I shall endeavor to show farther on, is not fully descriptive of the causes nor of morbid results.

The following classification is one which clinical experience, confirmed by autopsical examinations, has led me to adopt: Tubercular and Fibroid Phthisis.

FIRST CLASS, OR TUBERCULAR PHTHISIS.

*Two Divisions.*

First Division.—Uncomplicated Tubercular Lung, no adhesions.

Second Division. — Tubercular Lung, Adherent Pleuræ.

## SECOND CLASS, OR FIBROID PHTHISIS.

### *Two Divisions.*

First Division.—Adherent Pleuræ, with Fibroid Lung.

Second Division.—Adherent Pleuræ, with Tuberculated Fibroid Lung. Tuberculated fibroid lung is a term used to indicate cheesy degeneration, but not necessarily a result of tubercle.

This classification covers the whole ground—including accidents and complications.

### *First Class, First Division.*

Uncomplicated Tubercular Lung.—Tubercular concretions and cavities in the lung without adherent pleuræ or fibroid—sacculated tubercle—latent phthisis.

This form of phthisis is rare.

Louis says: "Nothing was so frequent as the adhesions of the lungs to the pleuræ, for in a hundred and twelve cases there only existed *one* in which the two lungs were free in the whole of their extent. We have only found the right lung completely without adhesions eight times; the left only seven, and in these cases there were either no tuberculous excavations, or only those of very limited dimensions."

Laennec and Louis include all those cases which are obscure in diagnosis, especially in the earlier stages, under the term latent phthisis. "These differences in the order and duration of the morbid phenomena do not interfere with the regular progress of the disease—do not, so to express ourselves, alter its physiognomy; but there are instances when its characters are so completely modified that its recognition is impossible before its progress is considerable: it is, in fact, *latent* for a longer or shorter period. At other times it assumes

the form and progress of acute diseases, its different periods seem confounded together, and the diagnosis is not less obscure than the opposite condition." *

The early history of the first division of tubercular phthisis is generally overlooked on account of the obscurity of the physical signs and symptoms, owing to the fact that there are no adhesions to convey the sounds of morbid changes in the lung into the chest-wall for easy recognition; the first observed evidences of the disease being those connected with the formation of a cavity.

Laennec says of latent phthisis: "It very seldom happens that phthisis is latent through its whole course; but it is by no means rare to meet with cases in which the characteristic symptoms show themselves only a few weeks, or even days, before death; and which had been previously mistaken for diseases of quite a different nature." †

These cases were evidently, according to our classification—Class First, Division First—uncomplicated tubercular phthisis, until "the characteristic symptoms" showed themselves "a few weeks or even days" before death, when they came under the second division of tubercular lung with adherent pleuræ. Laennec nor Louis knew anything about the applied laws of acoustics, nor did they know of residual air, and consequently they lacked the elementary knowledge for correct diagnostication of uncomplicated tubercular lung.

It is not wonderful that these early auscultators to whom we owe so much should have been unable to diagnosticate uncomplicated tubercular lung, for there were no adherent pleuræ for the ready conduction of sound

* Phthisis, by Louis. Chap, VIII., 372. Translated by Chas. Cowan, M.D., Washington, 1876.)

† Forbes's Translation, p. 327.

—telephoning, as it were, from the interior of the lung into the chest-wall. Even now the auscultator who does not recognize the diagnostic value of true respiratory murmur cannot appreciate the delicate but absolute sign of centric tubercular concretions nor of centric pneumonia, which is simply to comprehend the fullness or absence of true respiratory murmur, without which the evidence of the condition of the interior of the lung entirely escapes them.

The predisposing cause of uncomplicated tubercular consumption is a strong proclivity from inherited tendency. It occcurs most frequently in early adult life or in middle age, and its immediate cause is local or systemic irritation. Acute tuberculosis occurs in children at the periods of dentition, at puberty, and in middle life. The relation of acute tuberculosis to tubercular condition of the lungs, is not absolutely clear, but clinically children liable to head troubles in infancy, if they live to adult age, may have tubercular phthisis. Both in children and at adolescence the manifestations of tubercular invasions may occur in persons of full habit, with abundance of adipose.

At the first thought this seems incongruous, for tubercle is the feeblest of neoplasms and runs a rapid course of degeneration; but we must remember that adipose is not of itself a sign of strength, but it may exist at the period of tubercular invasion, connected with a marked prostration of vital power. Should a case be under observation before the appearance of cavities, it may be noticed that there is deficiency of true respiratory murmur, especially over the site of forming concretions, while at the same time there is slightly raised pitch under percussion. There are no rhonchi, râles, sibilous or sonorous, and possibly no cough. But just so soon as the nodules or encysted

tubercle begin to soften, there will be prostration, rise of temperature, quickened pulse and hurried breathing —perhaps cough and slight expectoration if the concretions should be near bronchial tubes, but when the abscess opens into a bronchus there may be expectoration of characteristic matter, and there may be fatal pneumorrhagia, depending upon the erosion of an artery occurring at the same time. Then for the first time the physical signs of a cavity are discoverable, but they are by no means so plain as when there are inter-pleural adhesions and fibroid lung. Healthy lung structure is a poor conductor of sound; but an attentive ear will discover a low note of amphoric character, especially in expiration. Should the cavity be large and connected with a large bronchus, there may be gurgling when it contains fluid. Coughing and expectoration are never excessive, as they may be in fibroid phthisis. Wasting and loss of weight commence to rapidly increase after the occurrence of cavities, as do also hectic, night-sweats, loss of appetite, etc. Louis gives two varieties of the latent form of phthisis. One rapid in its course, ending in a few weeks without any arrest in progress, while the other may linger and for a time give some hope of recovery. I have seen both varieties. One, in which there was an arrest of progress of disease in the lung, died with marked signs of meningeal tuberculosis.

Laennec also refers to *latent phthisis* and *acute phthisis*, but not in so clear a manner as Louis, and without detailing physical signs or post-mortem examinations. Except incidentally in one case, "a girl, eighteen years of age, who died in the hospital *Cochin*, without any emaciation, or other symptom except those of a severe feverish catarrh of less than a month's duration. Upon examining the body, the lungs were

found filled with tubercles more or less softened, of a size almost uniform, and none less than a filbert or almond." * This case was, no doubt, one of uncomplicated tubercular lung.

Rindfleish says: "That tuberculous phthisis is only a combination of scrofulous inflammation and tubercles" †

"Nodules as large as a pea, or even a walnut, are not uncommon." ‡

*Treatment.*—The early management of a case is in its prevention. Scrofulous diathesis indicates that the individual should be kept under the best hygienic influences, out-door exercise, pure air, and appropriate food, and that any local or systemic source of irritation should be removed. I consider chloride of ammonium as a preventive as well as a curative agent of very great value. It may be used in baths, by inhalation and by enema, as well as by the stomach. Dissolved in bay rum it is a pleasant sponge-bath with a flannel cloth night and morning. By inhalation in all the catarrhal conditions of the nasal and upper-air passages. By enema in threatened meningitis of children, and by the stomach in deep-seated "colds."

Should the disease have commenced, cod-liver oil, tonics, aids to digestion generally, change of air and scene in addition to hygienic conditions and chloride of ammonium may be beneficial. Also, digitalis sustains the action of the heart when enfeebled; atropia controls night-sweats; quinine and arsenic are anti-periodic, and may be adjuvant according to individual indications.

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* Laennec. Forbes's Trans., 4th edition, p. 328 and 329.

† Ziemssen, vol. V., p. 635, American edition.

‡ *Ib. id.* p. 642.

I have no doubt also that iron and iodine may be of great value in purifying and enriching the blood.

Recent excavations may be kept at rest, preventing extension of disease and of pneumorrhagia by strapping the affected side with elastic adhesive plasters.

Small blisters frequently applied over and around the region of excavations assist in arresting progress of disease.

Stimulants, when they promote sleep and digestion, should be taken at meals and at bed-time. Food should be abundant, easily digestible, varied, and moderately stimulating.

Forced expansion of the chest when nodules are softening, or after an excavation has been formed, must, of necessity, be avoided. But when the danger of hemorrhage has passed, it may be gradually resumed. A fatal hemorrhage rarely takes place after a cavity is a week old. Eroded arteries contract speedily.

Inhalations of medicated vapor may soothe irritation in the upper bronchiæ, prevent ulceration in the larynx and trachea, and may even reach excavations opening into large bronchiæ. A certain amount of medication may enter the system, especially chloride of ammonium, but we must remember that the residual air resists the entrance of irritating vapor into the true respiratory system; hence, there is generally disappointment where much benefit has been anticipated.

*Second Division of the First or Tubercular Class.*

Tubercular nodules and cavities following pleural adhesions.

The only difference of the second division of the tubercular class from the first is, that it commences with plastic exudation within the pleuræ—sacculated or nodular phthisis very soon following or becoming

evident. This division is larger than the uncomplicated tubercular, and is remarkable for the frequency in which it is terminated by fatal accidents, pneumorrhagia and hydropneumothorax. These accidents may occur in the first division as well as in the second or tuberculated division of fibroid phthisis, but in an experience of thirty years I do not remember a single case of fatal penumorrhagia occurring in any but in the second division of tubercular phthisis, at least none others were verified by post-mortem examinations.

In the first division of the first class the occurrence of tubercle is apparently spontaneous. If pleuritic adhesions afterward occur, they are accidental, and appear near the end of the disease; but in the second class adhesions precede and seem to excite tubercular deposits. I am fully aware that this fact cannot be fully appreciated except by those capable of recognizing the initial stage of inter-pleural plastic exudation.*

The immediate re-absorption of the plastic exudation may prevent tubercular deposits and its dangerous liabilities.

The following history in fatal cases usually obtains: Plastic exudation takes place within the pleuræ, over the upper half of the lungs, and tubercular concretions mostly centric are formed, and pass to the period of softening. Early in the disease one or more open into a bronchus, and if a branch of the pulmonary artery passing through the abscess opens at the same time, instantly blood will fill the air passages in that side of the chest, and, rising into the trachea, run over, filling the air passages in the other side of the chest—a few mouthfuls of blood are expectorated, when the mouth

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* See Dr. Brown-Séquard's "Archives of Scientific and Practical Medicine, March, 1873; the *Medical Record*, May 25, 1878."

and nose fill with frothy blood, the patient strangles—is literally drowned in a few minutes.*

The fatal occurrence of pneumorrhagia is always a surprise to the physician as well as to the patient and his friends, as the first indications of danger are only recognized when it is too late to interfere. The formation of tubercular nodules, centric, in otherwise healthy lungs, underneath adhesions and thickened pleuræ, cannot be easily diagnosticated, for there are no obvious physical signs except limited areas of dullness.

Post-mortem examinations show a few tubercular concretions, mostly central, near blood-vessels and bronchiæ, one or two of which have opened into a bronchus and into a branch of the pulmonary artery at the same time, and the bronchial tubes are filled with blood.

Should a softening nodule open into the pleuræ, leting in air and fluid, we would have hydropneumothorax. This accident occurs suddenly, causing great pain and dyspnœa. Sometimes the patient dies from the shock, or he may linger a few days; occasionally months. Some have recovered.

The diagnosis is easy. The sudden pain and dyspnœa direct attention to the affected side, and the tympanitic resonance under percussion, with amphoric respiration and metallic tinkling, are decisive.

Treatment will consist in immediately strapping the affected side with elastic adhesive plasters, relieving pain and controlling inflammation. If the amount of fluid escaping into the pleural cavity is small, the opening through the pleura may be closed, the fluid absorbed, and the patient live.

The consideration of both divisions of the first or

* Case viii., Physical Signs of Inter-pleural Pathological Processes. *Medical Record*, May 26, 1878.

tubercular class fully justifies the popular belief in the fatality of consumption. Fortunately the number is much less than that of the fibroid class, which is amenable to treatment.

### Second Class.—Fibroid Phthisis.

*Two Divisions.*

*First Division.—Adherent Pleuræ, with Fibroid Lung.*

This division represents a disease entirely opposed to that of the first division of the tubercular class.

In this there is loss of function only; in that necrosis of tissue, with loss of substance.

Many times, doubtless, pure fibroid has been mistaken for tuberculated fibroid phthisis, the second division of this class, on account of the gurgling, interpleural râles being misinterpreted as signs of cavernules in the lungs.

The physical signs of plastic exudation are soft, tearing, crepitant and sub-crepitant râles near the ear—not more than five or six lines distant—which are often present without any expectoration or cough, and which are heard in the same place from day to day.

If they were caused by mucus in the bronchial tubes, they would almost necessarily be accompanied by expectoration and cough; they would be at different distances from the ear—never so near, and would change their locality and quality at each examination.

I believe that nine-tenths of all forms of phthisis commence with inter-pleural plastic exudation, which is removable, when fresh, by proper management.

In consequence it is of the utmost importance that an early diagnosis should be made, in order that judicious but simple management, aided, if necessary, by positive treatment, may clear up all signs of the exudation,

and in accomplishing this, arrest the tendency to phthisis, diminishing the number of victims of the most common and the most fatal of diseases.

The inherited proclivities in fibroid phthisis are gout, gouty rheumatism and syphilis—factors of vital depression favorable to plastic exudation

But many times the proclivity is acquired, where the heredity is of health. Anxiety of mind, mental or vital depression long continued, may inaugurate a tendency to plastic exudation in the most healthful organization. Instances of a surviving husband or wife, after long watching at the bedside of one dying with phthisis, becoming consumptive are not unusual.

So frequent is this the case that the question of the transmissibility of phthisis has been mooted; but a conclusive answer is, that whatever may have been the character of the lingering disease of the first case, the second one resulting always begins with plastic exudation.

*Mental Depression.*—Students, men of exciting business, and lovers, especially when unsuccessful, are liable to inter-pleural exudation, which may be the beginning of phthisis. Soldiers after a defeat are liable to phthisis or typhoid fever.

*Vital Depression.*—Syphilis, or masturbation in those just arriving at adult age, small pox, or other of the exanthematous diseases, a badly managed pleurisy or pleuro-pneumonia, malaria, a wasting ulcer, a capital operation in surgery, may be followed by plastic exudation, which may end in consumption.

The depressing causes are so numerous that it is a wonder that these serious consequences from plastic exudations are not oftener observed. The exudation is no doubt much more frequent than we are aware, as many times it is immediately re-absorbed, and at other

times, although becoming organized, it may be of such limited extent, and so placed, as to remain innocuous during life. The exudation is a makeshift, as it were, of nature, and it is only when she is unable to remove it again that it becomes a source of inconvenience or of danger. If not re-absorbed, it becomes organized, and contracts according to natural law. The effect of which upon the pulmonary pleura is to contract it upon the air sacs immediately underneath, closing them and arresting their capillary circulation, which is then thrown back upon its two sources of pulmonary supply, that of the pulmonary artery and that of the bronchial, through the medium of the nutrient arteries. The obstruction to the circulation of the blood from the pulmonary artery is not of much importance, but that of the capillaries of the nutrient arteries seriously interferes with the circulation through the bronchial arteries. The nutrient arteries of the true respiratory system of the lungs are derived from the bronchial. They have no *venæ comites* to return their blood to the right heart for re-aëration, as all other arteries of the body have. The blood which they carry to the tissues of the true respiratory system for its nutrition is re-aërated as it passes through the capillaries into the radicles of the pulmonary vein—never becoming venous in character.*

This anatomical peculiarity is the key to many otherwise inexplicable phenomena of diseases of the lungs and of the pleuræ. It explains bronchorrhagia and bronchorrhœa. As before said, fibrination having taken place upon the pulmonary pleura, and contracting, the blood in the nutrient arteries is "back-watered," so to speak, upon the bronchial, whose natural relief is trans-

* "Respiratory Murmurs," read before the N. Y. Academy of Medicine, 1872.

fusion through the mucous membrane, of blood, fibrine, serum or mucus.

Consequently the indications are that the bronchorrhagia or bronchorrhœa following should be treated as effect, and not as disease. They are the natural results of the capillary obstruction. Such bronchorrhœa is different from primary catarrh, inasmuch as its primary cause is not in the mucous membrane, but far removed from it. Also fibrination within the pleuræ alone is not pneumonia, as has been mistakenly diagnosticated.

A careful physical examination will show that at this stage all the changes that have taken place are within the pleuræ. For these pregnant reasons I cannot accept the term catarrhal pneumonia as descriptive of its pathological processes. All of these signs and conditions are the accumulating results of obstruction of the capillaries immediately subtending the pulmonary pleuræ.

From time to time fibrination progresses induced by slight causes, until the patient yields to the crippling process of contraction, stoops forward, with hurried breathing and spasmodic cough. Old adhesions are reinforced by new exudation caused by colds, fatigue, emotion or "worry."

The second stage of the first division now commences, when the inflammatory process begins to extend into and through the lung itself, and portion after portion of the true respiratory system becomes involved in the contracting fibroid. The heart and lungs are displaced upwards, downwards or sideways, or are bound to the chest-wall. Cardiac murmurs result which may deceive the physician into making an error in diagnosis of heart disease. The heart struggles, palpitates, sometimes hypertrophies or dilates, and fails to properly carry on

the circulation; stasis, increased fibrination, continually recurring, spasmodic, strangling, almost suffocating cough, fill up a picture of a pitiable condition. Autopsies confirm the diagnosis in a remarkable manner. Adhesions within the pleuræ fasten the lung to the chest-wall, sometimes to the mediastinum, the pericardial sac to the lungs, and all are drawn out of their normal position until the apex of the heart has been found on a level with the lower border of the fourth rib.*

The earliest physical signs of fibroid are simply those of plastic exudation within the pleuræ. The percussion note is slightly flat, and raised in pitch as if parchment or paper were spread over the chest-wall. The râles are fine, soft, moist, tearing. It requires a practised ear sometimes to discover these delicate signs, but even a beginner in auscultation will notice that the respiration is harsher over some one region of the affected chest than another; let him fix his attention in listening to this rough respiration, and fill his own lungs at the same time and in the same way as does the patient, and after a little while he will be able to analyze this roughness, and find that it is made up of innumerable moist, soft râles, very fine and very frequent. At the same time he may hear the true respiratory murmur, when it exists, just beyond the inter-pleural râles, with as much certainty in measuring the distance as he could do it by sight, welling up like the distant roar of the sea. When he finally hears these râles and distinguishes at the same time the true respiratory murmur, he will be convinced of two important facts, that there is lymph exudation within the pleuræ and that the lungs are free. In time, these soft, almost unrecognizable râles

* Case IV., Phys. Signs of In. Pl. Path. Process. The *Medical Record*, May 15th, 1878.

become more distinct, even dry and crackling, and then all doubt of their existence is cleared up.

There may be an abundance of râles with neither cough nor expectoration; but unless the exudation is re-absorbed each will begin in time; at first a short cough with viscid mucus, colorless or slightly tinged with blood, but afterwards becoming more expulsive with profuse expectoration and assuming a greenish hue.

The dyspnœa is frequently out of all proportion to the amount of pathological results in the pleuræ or of the congestion of the lungs.

If the serious mistake has been made of considering the early signs of plastic exudation as those of catarrh or of bronchitis only, strong adhesions may result and become a point of irritation, which may continually induce new exudation and increased disability

The physical signs of firm adhesions are greater flatness under percussion, and perhaps a shade of dullness over areas of thickened pleura or of condensation of lung, with a great variety of râles, fine, dry, moist, coarse, or a combination of all of these. The rational signs are distressing dyspnœa; spasmodic coughing, with copious expectoration; irregular palpitation of the heart; temperature varying from natural to 38.9° C. 40°C.; variable appetite; sometimes sleeping quietly when lying down; in other cases catching what sleep they can in an arm-chair, or sitting up and leaning forward in bed; progressive emaciation and debility, until a new cold, greater hyperæmia, fresh exudation, and the life is closed out. Louis notes that in autopsies it was found that fresh plastic exudation, occurring in the last days of exhausted vitality, was evidence of debility. No doubt it is so at the commencement as well as at the end in phthisis cases.

*Treatment* of first division of the fibroid class is an easy problem at the beginning, but grows more difficult every day of its after existence. Organization may take place very soon after exudation, but generally appropriate management will cause its speedy removal. Even when the exudation is some weeks or months old, *positive* treatment will soon clear up the evidences of disability and disease. Regulated or systematic expansion of the chest in the open air, with appropriate food, are of the first importance. Walking, or riding on horseback, in the country, and habitually filling the lungs and holding in the breath, a little more and a little longer than usual, with milk diet in abundance, is generally sufficient in recent exudation without medication.

*Case* I.—Rev. —— ——, 34 years old, born in New Jersey; father died at the age of 54 of phthisis; family history otherwise good. During the great heat of the summer of 1878 ministerial duties were heavy, was de pressed about business affairs, and began to be ill. After feeling weak and "out of sorts" for some time, was taken with hæmoptysis on the morning of July 13, 1878. Became apprehensive, sleepless, could eat, but had no appetite; fell in weight from 122 to 117 pounds. Hawked up mucus, but had no cough proper. When lying down could hear whirring noises in chest. Had stitches mostly in left side about the heart, with palpitation. Physical examination discovered a few distinct râles over right lung; left side a few râles at upper part, but in the lower part an abundance of fine, subcrepitant râles back and front. Respiration feeble; could not fill the chest fully in inspiration; no dullness, but a little flatness under percussion in lower part of left side.

*Diagnosis.*—Plastic exudation within the pleuræ,

mostly in the lower part of the left side. Directed systematic expansion of chest in open air, walking, milk diet. Took no medicine, except cod-liver oil; rubbed down with English glove night and morning.

Re-examined Nov. 7, 1878. Respiration and expansion improved, but râles remain.

Re-examined March 1, 1879. All signs of exudation have disappeared. Allowed to return to his ministerial duties. Weight, 130 pounds. Eats well; sleeps well, unless excited, and feels well. Walks five or six miles every afternoon, in addition to out-door exercise in the morning; has walked ten or twelve miles in a day without over-fatigue. Chest was measured on the 16th of November last, and again first of April; under the arms and under nipple. Gained under the arms, after exhausting the lungs, half an inch, in ordinary respiration three-fourths of an inch, and one inch and one-fourth after full inspiration. Under nipple gained half an inch in forced expiration, one-and-three-fourths inches in ordinary respiration, and two inches in full inspiration.*

With mild medication the time of recovery may be shortened, and its use is advisable if there is doubt about the organization of the exudation.

*Case* II.—D. E. returned from Florida in the spring of 1878. Took cold about two months before leaving the South; continued to cough, rapidly lost weight, from 180 to 160 lbs.; had two attacks of hæmoptysis. Physical examination discovered subcrepitous râles right side posteriorly; appetite poor; dyspnœa on exertion. Advised to go to Harper's Ferry, Va., and commence walking eight to fifteen miles each day, syste-

* It is seven years since the first examination, and he has had no return of chest disease.

matically expanding the chest, and living on milk diet, and in addition to take a cold infusion of wild cherry bark with chloride of ammonium—two ounces of the bark and one of ammonium in two pints of cold water; tablespoonful about every hour. This was done strictly, and he returned in about three weeks. All signs of plastic exudation had disappeared; had regained the weight he had lost; had no cough, no dyspnœa in exercise, and has remained well since.

But should the system be in no condition to respond to those simple measures, or if the organization of the exudation has resulted in firm adhesions of the pleuræ, with commencing consolidation of the lung, and the simple means fail, it may be necessary to resort to positive medication by mercurials—calomel and Dover's powder in small doses until the teeth are tender, which may be followed by bichloride of mercury in Huxham's tincture of bark in small doses, and may be continued for months in addition to the chloride of ammonium, and systematic expansion of the chest in the open air, milk diet, etc.

*Case* III.—A. R., native of Scotland, 39 years of age, clerk. Family history good. Weight in health, 165 lbs. Began to be ill in 1874. Frequently took colds; had "catarrh," but kept at business; gradually grew worse. In 1875 had some inflammation of the chest, which was checked; had severe coughing spells, with loss of strength and short breath; all symptoms growing gradually worse until October, 1878, when he came to be examined.

Pulse frequent and irritable; breathing hurried; constant coughing; expectorating yellowish thick mucus; appetite poor; disturbed sleep; weighed 130 lbs.

*Physical examination.*—Almost no expansion in right side; restricted on left; dullness over right lung,

especially over middle portion; not so great over left; fine dry râles over right side, especially over middle portion; some crackling râles at summit of right lung; softer tearing râles over left side.

*Diagnosis.*—Extensive adhesions in both pleuræ; old and organized in the right, with consolidation of middle portion of lung; fibroid phthisis, second stage.

Placed him at once on calomel and Dover's powder, to make the teeth slightly sore; then to follow with chloride of ammonium and wild cherry bark, cold infusion, and frequent small blisters; systematic expansion of the chest in the open air, freedom from business, milk diet, etc. The mercurial treatment was resumed three times, and carried almost to the point of mercurialization, followed by blisters, etc., with marked improvement of rational and physical signs; each time chloride of ammonium and wild cherry bark, with bichloride of mercury. One thirty-second of a grain three times daily in a compound tincture of Peruvian bark were continued afterwards.

He was permitted to return to his business in January.

*Re-examined April* 22, 1879.—Has gained twenty pounds in weight since October last. Has no cough; pulse natural; respiration quiet; temperature, 37° C. (98.6 F.).

Physical examination shows increased expansion of chest; no dullness; a little flatness; some thickened pleuræ still remains over middle portion of right lung behind; no râles on either side.

Has not regained full strength, although very much improved; a little short breathed on severe exertion; eats well, sleeps well, and feels perfectly well when not over exercising, and has had no return of chest disease.

When the fibroid is extensive both in the pleuræ and

in the lung, as in the above case, mercurialization nearly to the point of salivation may be absolutely necessary to relieve the patient. The result in Case III. was exceptionally favorable, and cannot be regarded as the rule for all cases of fibroid in the second stage. Yet to save one such case from among a number is very encouraging. The careful physician, who knows how to use his tools, will have no fear of doing injury. He will carry the use of this powerful remedy just so far as is necessary to accomplish the desired end, and no further. The blister will be most efficient when the system is under the influence of the mercurial.

Systematic expansion of the chest must not for one moment be lost sight of, no matter what form of medication may be adopted. Indeed it should be considered that all medication is auxiliary to expansion—to make expansion possible.

Gently filling the lungs, holding the breath, depending upon the rarefaction of the cool, inspired air after mixing with the heated, residual air, to dilate the lungs and gain expansion of the chest. When there is no irritation of the lungs or pleuræ the air may be forced into the lungs and held as long as possible, that contracting adhesions may be overcome.*

Accurate measurements of the chest should be made and recorded at intervals, that progress may be ascertained and patient encouraged. Perhaps no simple method of gradual expansion is more effectual than riding on a fast walking horse. The instinctive balancing of one's self on the horse in the rolling motion of fast walking keeps the chest expanded, and systematically

*The Pneumatic Cabinet may be of great advantage in expanding the chest where the adhesions are firmly organized, and after systematic expansion by breathing exercises have been successfully put in operation.

exercises all the muscles of the body without overfatigue. In forcible expansion care must be taken not to do harm. Adhesions must not be torn nor put violently upon the stretch, or the result may be extension of inflammatory action and further disability by new exudation. The pleura has been torn from the adherent lung by the accident of falling, and death has resulted from hemorrhage resembling pulmonary apoplexy.

In connection with systematic expansion the subject of climate is important, as expansion in pure air is more beneficial than in bad air. Change of scene and of accustomed thought is desirable, as also out-door exercise and cheerful amusement with a congenial friend in a cool, equable climate free from malaria, in balsamic forests. But even then change should again be made. The patient does best who goes from place to place. The influence of change upon the digestive organs is a matter of common observation. Sea voyages for those living inland, to the mountains for those living by the sea, even from a good to a poor climate may give a temporary benefit. I have known patients to improve rapidly by coming from healthy hill countries to New York, which certainly cannot boast of perfect climate for a phthisis patient.

Any one locality, however good, should not be recommended for all. One whose taste runs in that direction will do best where there is hardship and roughing it, with plenty of incident, while others, and especially women, may do better in congenial society, surrounded by the elegancies and comforts of fashionable life.

We have on our continent every variety of climate and scene, California, Colorado, Minnesota, Canada, Texas, Florida, North and South Carolina, Georgia and Virginia, or the Adirondacks. Short voyages also take us to the Bermudas and West India Islands.

But if there is progression in fibrination, the time may come when the patient must desist from exercise, and keep his room or even his bed for a lengthened period, using the gentlest means to keep the chest expanded, living upon the most nutritious and stimulating food. Using rectal alimentation with defibrinated blood, intelligent mercurialization, blistering, and tonics to cause re-absorption of newly exuded matter which may so free the lungs again that out-door gentle exercise may be resumed when summer has set in. From the latter part of February until the first week in June a phthisis patient who cannot seek a better climate should keep his room by a cheerful fire, and take only such exercise as he can indoors.

### Second Division of Fibroid, or Second Class.

### *Tuberculated Fibroid Phthisis.*

To this division belong the great majority of the cases of phthisis which come under our observation, too late for curative treatment.

The disease is essentially fibroid; the tubercular element is a complication, and is accidental. Niemeyer says that the fear in a case of catarrhal pneumonia is that it may become tubercular. Substituting the term fibroid for that of catarrhal pneumonia, I would entirely agree with his anxiety in regard to this complication. The lowered vital power in a fibroid lung or pleura, with the constant irritation caused by the inter-pleural adhesions, invite the exudation of tubercle. A scrofulous diathesis with fibroid lung is almost certain to become tuberculated, and it is this fact which makes it so necessary to watch and to remove the first beginnings of the fibroid condition.

The causes, history, physical signs and treatment of

this division up to the time of tuberculation have already been glanced at in the consideration of the first division of the fibroid class. The new physical signs denoting the advent of tuberculation will need to be watched for with great assiduity, for upon their appearance or non-appearance depends very largely the hope or despair which will govern the efforts for cure or for palliation.

These signs are areas of dullness with raised pitch under percussion, with loss of true respiratory murmur, followed by bronchial breathing, bronchophony, raised temperature, hurried pulse, and respiration. Decided exacerbations, chill, fever and sweating, periodically returning. The cold sweat coming on after midnight is like the approach of death, and is horrible to the patient. When the tubercular masses soften and open into a bronchus, the characteristic expectoration may announce the formation of a cavity, or the expectoration may not be observed. A general amelioration of all the symptoms may occur at this period. The chills and fever may subside, the pulse and temperature may fall to normal, the respiration become slower and fuller, the hectic and night sweat disappear. Perhaps the patient begins to eat and sleep well, and from this time forward there may be continuous improvement, but it is unusual.

Great injustice may happen to the attendant physician should he be changed for another a short time before the formation of a cavity, for the great improvement of all the symptoms will naturally be attributed to the new doctor. Many patent medicines have gained great popularity from having been "*tried*" in the right time. But, unfortunately for the patient, such complete relief is rarely obtained. Other tuberculations may be going through the same process of soften-

ing, and the amelioration may be but partial and only for a short time.

The physical signs of a cavity are made exceedingly plain by the good sound-conducting quality of fibroid lung and adherent pleuræ. The cavernous or amphoric respiration, and the reverberations or echoes of râles and gurgles in the cavities with pectoriloquy, vocal and whispering, leave no doubt of what has taken place.

Auscultation may discover remaining concretions which may soften in time and repeat the same signs and symptoms until they also are discharged.

The condition of cavities may be studied for the benefit of the patient: As to whether they are empty or filled or partly filled with fluid. Also as to the manner of their opening into a bronchus, from the walls of the cavity or from the roof or from the floor. Should the opening be from the bottom of the cavity, it will always be empty when the patient is in an upright position. Should it be from the top of the cavity it may be overlooked during examinations made in the middle of the day, the usual time of visits, but may be readily discovered early in the morning, or after the patient has retired in the evening, times when the cavity will be partly empty from the recumbency of the patient.

A knowledge of these simple facts, gained by careful auscultation, may be utilized for the comfort of the sufferer.

Learning the *manner* of the connection with a bronchus may enable us to relieve distressing night-cough without the use of opiates. A patient may sleep quietly after retiring, for some hours, and then be awakened and kept awake by cough the rest of the night, or he may commence the moment he lies down or turns upon one side, and he instinctively seeks the

position which gives him most ease from strangling cough, and submits to a constant teasing cough that only yields to large doses of opium. After examination, teach him to take that position which will soonest empty the cavity and keep it, notwithstanding the coughing, until the cavity is thoroughly emptied, then he can take his usual position and sleep quietly until morning.

These practical facts were embodied in a paper prepared for the Academy of Medicine, by the late Dr. Geo. P. Cammann, and which I had the honor of reading before the Academy after the writer's death.

Cavities in the lungs are not always of a tuberculous origin. A portion of lung tissue may necrose from strangulation by contracting fibroid and become gangrenous, and a cavity result which may remain open, or even enlarge by wasting from its walls. It may be of traumatic origin. I have known one to occur from tapping with a trocar into a lung bound to the chest-walls by adhesions. A ball of lead has been the cause of a cavity after having been in the lung for many years. From whatever cause, a cavity in the lung is a grave accident.

Dry, crackling râles from old pleuritic adhesions are loudly echoed in a cavity near the surface of the lung, and assist in differentiating it from a dilated bronchus, in which they are much feebler, if heard at all, and the sound seems to escape, while in a cavity they are defined and echoed from the walls.

Cracked-pot sound is also easily distinguished when the cavity is near the surface, but even when centric the expert ear may catch the peculiarity of the double-echoed quality of sound with that of the sudden expulsion of air into the bronchus.

Treatment of the tuberculated division of the second

class must be a judicious combination of that already given for fibroid and for purely tubercular, with the hope of delaying progress, if not arresting it altogether.

The earliest signs of plastic exudation within the pleuræ must be heeded and removed is the lesson that the consideration of this formidable disease impresses upon us, but if the fibroid lung has become tuberculated, there must be a double endeavor to prevent the extension both of fibroid and of the tubercular. The resort to mercurials must be more sparingly made than in the purely fibroid, and yet they must not be wholly disused. The bichloride of mercury, with tonics, will be the principal resort. Chloride of ammonium will be of more value than in either the pure tubercular or fibroid alone, as it meets the indications in both. The exercise must be adapted to the conditions, and too forcible expansion must not be made. Milk diet in large quantities must be encouraged and insisted upon.

Lord Bacon says, in effect, that many believe they cannot take milk without becoming bilious, because they take but little at a time, which coagulates, but that if they take large draughts, the acid is diluted, and digestion will take place. I have repeatedly demonstrated the truth of his observation. In order to take large quantities of milk, it is necessary to omit other kinds of animal food. Two or three quarts of good milk may be taken daily for weeks, even by a feeble person. The stomach must be educated to receive this quantity, and it must be done gradually. In fibroid phthisis the patients are apt to be carnivorous, and have contracted stomachs, so that at first they are unable to take a large amount of food at one time. But system and perseverance will overcome this difficulty. By the constant use of milk the stomach dilates, and the blood-vessels enlarge, and more nutrition is

carried to the capillaries, and weight of the body will be increased.

The increase in weight, which comes to drinkers of large quantities of any liquid, is owing to this acquired capacity to receive nutrition. Large quantities of milk at regular intervals, with systematic expansion of the chest, stands first in importance in treatment of all forms of fibroid phthisis. The increasing deposit of fat in the system is an assurance that phthisis is held in abeyance. Occasionally a change may be made, and a mixed diet of more stimulating food may be allowed, to continue only for a short time, again to return to strict milk diet, until health is restored.

The subject of tubercle I have not attempted to discuss, and the same may be said of minute pathology and histology, except in a clinical and practical way, leaving the niceties to be settled by those who are making them a subject of particular study.

NOTE.—The above article is but an outline sketch which I hope to see elaborated and filled out.

## ARTICLE IX.

### Is Consumption Communicable?*

From the earlier days of medicine to the present time there has ever been a popular belief that consumption is communicable. Such a widespread and general opinion, continuing for ages and in many countries, must have had some foundation in fact. Cases of consumption have followed each other under circumstances which have impressed observers as proof of their infectious character. As when a husband or wife who has watched with the deepest solicitude the long-continued and vacillating illness of the other, is finally overwhelmed with grief at the fatal result, sickens and dies under similar conditions.

The profession has at times inclined to the popular faith, and again has rejected it.

The discovery of true tubercle by Bayle in 1804, and of the methods and value of auscultation by Laennec, published in 1819, threw new light upon all diseases included under the common name of consumption. It did more, unfortunately—it filled the professional mind with the idea of tubercle, to the exclusion of other and common forms of consumptive diseases.

The very important doctrines taught by Broussais, in Laennec's time, because they were not all of tubercle, were overshadowed, obscured, and misunderstood. The immense advantage of physical diagnosis by auscultation and percussion in getting a true mental picture of the pathological conditions of the chest was

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* *New York Medical Journal*, December 1, 1883.

certainly weakened by the adoption of the exclusive doctrine of tuberculosis.

The erroneous interpretation of the respiratory act and of the significance of its murmurs, as taught by Laennec and his followers, confirmed them in the pathological error that all forms of consumption must necessarily be tuberculous. But careful post-mortem examinations grew in favor, and the microscope vastly extended knowledge of pathological results, and has established the belief with many that the tubercular is but one and not the most common form of phthisis. Many are still groping among the débris of protoplasm, cells, and proliferation, anxiously searching for the specific evidence of tuberculosis as an entity self-existing and self-propagating—something which has a separate life from the life of the body, which is independent of it, antagonistic to it, which overcomes it.

It is said that the giant cell characterizes tubercle and the spindle-shaped cancer, and that by them we are able to distinguish tubercular and cancerous products. But this knowledge of them does not determine the life-producing origin of tubercle nor that of cancer; whether they have a distinct life outside the life of the body, and have only an accidental connection with it, or whether these morbid cell-forms are merely the materialized expression of disease-action of the immaterial life of the body. Animals have been experimented upon by inoculation of tuberculous matter, and tubercle has been the result, and it has been claimed that the question was solved in the affirmative. But, again, animals were inoculated with non-tuberculous matter, and the result was tubercle, proving that the character of the inoculated matter had nothing to do with the tuberculated results, but

that irritation was the cause, and the result would be tubercle or cancer, according to the inherent tendency of the individual either to tubercle or to cancer. The irritation of teething endangers tubercular meningitis in children, and phthisis may result from the irritation of adhesions of the pleura. Had not this theory of tubercular inoculation disestablished itself by these experiments, it would still remain that inoculation is not infection, that poisoning the system by inoculation by any *materies morbi* is not conveying a germinating parasite into healthful respiratory organs, and producing disease in them of its own kind. But lately the medical world has been set wild by the publication of the discovery of bacilli in tubercular cavities and in tubercular sputa.

It has been shown, too, by experiment that these independent life-forms may propagate themselves outside the body and in other menstrua than the débris of decaying tubercular cavities.

These facts are demonstrated by other careful observers, and it seems to me that they open the door to investigation which will ultimately clear the subject of a vast deal of superfluous error. But the deductions of Professor Koch are that these self-producing life-forms are the cause of tuberculosis and of tubercle, and propagate their kind in healthful human lung, and, that their germs taking wings, are carried to and transplanted in other healthful lungs, and are the cause of other cases of phthisis.

The propagation of these parasites being rapid and abundant, and the medium of germ conveyance the air we breathe, the danger therefrom becomes appalling to fearful minds, who dread the ravages of this most deadly of human diseases. To be entirely consistent, the germ theorists must deny the influence of heredity

and external conditions, of local irritations or the depression of vital dynamics, as causes of consumption.

If it were not for the adoption of Professor Koch's theories, as well as the acknowledgment of his discovery of bacilli by gentlemen of high scientific attainments, such as Professor Rühle, of Bonn, and others, controversy would be unnecessary; but, as it is, we must examine the subject critically but dispassionately.

Fibroid phthisis is not included in the form of consumption claimed to be propagated by bacilli. The germ theorists assume that all forms of phthisis are tubercular. But a large number of cases are fibroid, pure and simple, in which the diathesis is gouty or rheumatic, and not scrofulous. These ought to be exempt from suspicion even. Again, a vast majority of cases of tuberculated phthisis, that is with excavations, commence with plastic exudation within the pleuræ. These, on account of bronchial complications, are called by Niemeyer "catarrhal pneumonia," and he says "the great fear is that they may become tubercular." This fear is born of experience, and should direct us to proceed energetically, at the same time judiciously, to remove the plastic exudation while it is easy of accomplishment. It may be well to state here that we make a distinction between tuberculosis and tuberculated phthisis. Tuberculosis is the systemic disease which gives birth to true tubercle—the miliary tubercle of Bayle. Tuberculated phthisis is a term used for convenience and denotes the result of cheesy degeneration, in which cavities take place as a result of tuberculosis or other causes of necrosis of the lungs.

The number of uncomplicated cases of tubercular phthisis—that is, of tubercle forming into concretions or nodules and being encapsulated, with no pleuritic adhesions and without fibroid in the lung, is extremely

small. In a practice of more than thirty years in dispensary, hospital, and private, I cannot remember more than a very few cases. Laennec and Louis evidently refer to these cases under the term of latent phthisis and acute phthisis.

This small number, commencing centrally in the lungs and not involving the pleura, are the only ones which could have had a parasitic origin. But even in these it is doubtful whether bacilli had anything to do.

I do not doubt the discovery of bacilli in great abundance in tuberculous cavities and in the sputa of tubercular consumptives, as well as in the adjacent tissues, but I cannot accept the inference that they are the essential causes of tubercle. They may find in a tuberculous cavity a fit soil or home where they may grow and multiply. There may be spores, eggs, germs, laid there by their parents, which, when perfected, may fly away to seek other tuberculous cavities in which to lay their eggs, etc.

Is there not analogy in the green-bottle fly that seeks carrion in which to lay its eggs, which, hatched into maggots, may increase the rapidity of the destruction of the carrion during their growth, but, becoming full-grown, fly away to seek other carrion in which to plant their eggs, and thus continually propagate their race?

But the bacillus is not necessary to explain the occurrence, cause, and course of phthisis—fibroid or tubercular. As has been stated, all but a very small number of cases commence as fibroid—that is, with plastic exudation within the pleura, in which the bacillus is not a factor. This primary condition of phthisis may be the result of depressed vital power from various causes, long-continued and violent emotion, anxiety, worry, grief, or disappointment, more than from catarrhal causes. Or it may, but in a less degree, be

the result of adhesions from acute pleurisy, which are a physical cause of vital depression.

A mother, after watching her children, three or four in number, through scarlatina of a severe type, began to cough, lose weight, and finally died of phthisis. She was well when the children were taken ill; she was a loving, anxious mother, and as they were attacked successively the time of her anxiety was prolonged. The children all recovered, but the mother was sacrificed. She was not aware of having taken cold. The cough was so insidious that no one could tell when it commenced. Had there been the same prolonged anxiety over a case of phthisis, followed by inconsolable despair at the loss of the loved one, it would have seemed to prove the communicability of consumption. But scarlatina germs do not originate phthisis, nor do bacilli.

Even in the ordinary forms of tubercular phthisis, for a considerable time the disease is simply fibroid—preventable phthisis.

One word for the poor consumptives. Morbidly sensitive to all unpleasant sights, smells, and surroundings, whose greatest comfort is in kind and sympathizing companionship, is it not the refinement of cruelty to drive away from them unnecessarily those who should minister to their suffering as is done by the false notion of tubercular infection.

*Quotations from Current Literature in regard to Bacilli, with Notes by D. M. Cammann, M. D.*

On March 24, 1882, Dr. Robert Koch communicated to the Physiological Society of Berlin the result of a series of elaborate investigations into the etiology of tuberculosis.

He believes tuberculosis to be caused by a parasite,

the parasite being a bacillus and being distinguished from other bacilli by its behavior towards the coloring agent "vesuvin." The tubercle bacillus is slender, rod-shaped, about five times as long as it is broad, and varying in length from one quarter to the whole diameter of a red blood corpuscle. The method pursued in finding the bacillus was as follows: "The tuberculous substance was either spread out upon a cover-glass, and dried and exposed to heat, or a piece of tuberculous organ was placed in alcohol, and afterwards cut into fine sections. A particular solution of methylene-blue was made, a weak solution of potash being added, the cover-glass coated with tuberculous matter (or a section of the organ) was then placed in the solution for twenty or twenty-four hours, but half an hour sufficed if the solution were warmed in a water-bath up to 40° C. The cover-glass, which comes out a deep blue, is then treated with a concentrated watery solution of 'vesuvin' for one or two minutes, and is afterwards washed with distilled water. The blue of the mythelene has visibly changed to brown; under the microscope all the amorphous detritus and fragments of tissue spread out on the glass are brown, *but the tubercle bacteria remain blue.*" — *Braithwaite's Retrospect*, July, 1882.

The bacillus was oftenest found in the interior of giant cells. Not every giant cell or group of cells contained it, but those which were free were old cells which had once held bacilli and had gotten rid of them. They may become few or disappear entirely. They are usually found in large numbers in cavities. To show that the bacillus is the cause, and not a mere accompaniment, of tuberculosis, Koch proceeded to separate it from other substances by a series of "cultivations." He took the blood-plasma of the ox or the sheep, and

after repeated applications of heat, he boiled it to a coagulum, "at the same time inclining the test-tube so that the coagulum might cover a considerable surface. It was on this nutrient soil that he proposed to 'grow' the tubercle-bacillus without the intervention of moisture." After taking a piece of tuberculous substance—usually from the lung of the ape or of man—and carefully washing it several times in a solution of corrosive sublimate, the outer layer was removed, and from within was taken a portion "into which it was to be expected that no bacteria of putrefaction had penetrated." The piece of tuberculous substance was then broken up and thrown over the surface of the coagulum, and the test tube kept at a uniform temperature of 37° to 38°C. If during the first week any activity showed itself, it was supposed that the bacteria of putrefaction were present, and the experiment was not continued. Usually about the tenth day could be seen on the surface of the coagulum "a number of very small points or dry-looking scales which surrounded the pieces of tubercle that had been laid out, in circuits more or less wide, according to the extent of breaking up or dispersion of the tubercle fragments at the time when they were sown." These dry scales were taken to be colonies of the bacillus. After a few weeks the scales cease to enlarge, and they are transferred on heated platinum wire to another test-tube prepared in a similar manner. This series of "cultivations" is continued through ten or a dozen times, and for a period of four or five months. With these dry scales numerous animals were inoculated, and without a single exception all the inoculated animals acquired tuberculosis, the tubercles having the structure of the original tubercle. Dr. Koch claims that these results are due to the introduction of the bacillus per se.

Since Koch announced his discovery his experiments have been repeated by several observers. That the bacillus is, as a rule, found in the sputa of persons having tubercular phthisis is confirmed by Ziehl, Fraentzel and Balmer, Belfield, Hierchfelder, and many others. Dr. Spina, of Vienna, while agreeing with Koch in always finding bacilli in the sputa, denies that they occur constantly in the tuberculous organs of man. He could never find them in the tubercles, which stood in no connection with the open air, and he concludes by saying that "the bacilli of tuberculosis are the result, not the cause, of the disease."

Cases of miliary tuberculosis are recorded by Pruden (*Med. Record*, June 16, 1883, p. 645) in which "no baccilli could be detected by the most exhaustive search." Considerable evidence is available to show that the bacillus is less frequently found in tubercle tissue than in the sputa of phthisical persons, and that in the former it is chiefly found in the walls and contents of cavities, and in cheesy areas, especially in those that are disintegrating.

## ARTICLE X.

### Bronchitis.

Bronchitis may be divided into three varieties.

1st. Simple, or catarrhal, affecting only the bronchial mucous membranes; is always acute and self limiting; not extending over two weeks in time. The rise of temperature is but little, frequently none at all. It is popularly considered "only a cold."

2d. Severe, or inflammatory, affecting both the mucous membrane and the fibrous sheath. The temperature may run high; it may be irregular in its continuance, and be of serious importance, frequently complicated, or complicating pneumonia and pleurisy.

3d. If occurring in the course of pneumonia, during convalescence it may prove surprisingly and speedily fatal. The inflammatory results may extend beyond the fibrous sheath into the peri-bronchial spaces, —peri-bronchitis. It is frequently, if not always, complicated with plastic exudation within the pleuræ.

4th. It is called chronic bronchitis when it becomes peri-bronchial or inter-pleural in its complications, for then the inflammatory and plastic conditions have a tendency to constantly recur and the plastic pathological products are more or less permanent.

### Catarrhal Bronchitis.

Uncomplicated, this disease affects only the bronchial mucous membrane. Its causes are sudden changes of temperature from hot to cold, or from cold to hot, or exposures to wind, or dampness with insufficient clothing. Or it may occur from local irritation of the

mucous membrane, as from dust or other extraneous matter, or it may be from irritating gases. Its site is the mucous membrane of the tidal air passages. It does not extend into the true respiratory system, which is constantly occupied by the residual air. Its limitation is anatomical. It only affects the mucous membrane supplied by the superficial bronchial arteries.

It has but little, if any rise of temperature, and is unaccompanied by constitutional symptoms. It does not affect the appetite nor digestion.

It is sometimes epidemic in its character, affecting mostly the mucous membrane of the air passages of the nose, pharynx and larynx. It is called influenza or grippe, in which case it differs from catarrh from ordinary causes. Catarrh is only the more prominent symptom of an epidemic disease affecting the organic life of the body.

### Physical Signs.

There are two stages of simple catarrhal bronchitis. First or dry stage, in which there is no secretion; the broncho-respiratory murmur is harsh in character and somewhat raised in pitch. It can be heard everywhere over the chest, but with greatest distinctness in the neck and in the upper part of the chest.

There are no râles, that is, there are no interrupted noises like tearing of cloth or paper. There are sometimes sonorous and sibillant rhonchi, continuous sounds, but these are adventitious and are confined to the second stage.

The broncho-respiratory murmur of the first or dry stage is a dry murmur whenever it is heard. It is loud, harsh and near the ear in the neck and clavicular region. It is consonated in the true respiratory system. The vibrations formed by the air-and-tube fric-

tion, the tidal air, pass downwards through the columns of static air in the convective tubes and are delivered through the air-sacs into the chest wall as through a speaking tube. It alters and obscures the normal broncho-respiratory murmur, for it is harsher, drier and raised in pitch.

In the second stage the breath sounds become moister in character. When mucus collects in the upper passages in sufficient quantity to be moved backwards and forwards by the tidal air, there will be mucous râles, —always large, and heard over different parts of the chest, having no points of greatest intensity except there be pleuritic adhesions to convey them into the chest wall.

These mucous râles, also, are intermittent, for the accumulation moves backwards and forwards only a few times before it is loosened sufficiently to be expectorated, when the râles cease. But in a short time the mucus collects again, and the râles reappear. They are always distant from the ear unless brought directly to it by conduction. They are always distinguishable from similar râles heard from inter-pleural causes. Inter-pleural râles are heard only over the site of their formation. Inter-bronchial over a large space, if not over the whole lung. Inter-bronchial or true mucous râles are intermittent, and soon change or pass away. They can scarcely be distinguished from mucous râles in the nasal air-passages, as both are consonated in the true respiratory system and are heard over a large space. But this can be done by auscultating the neck with a stethoscope. If they are nasal or pharyngeal, or laryngeal, they will be heard in the neck, but not if they are inter-bronchial.

## Treatment of Simple Catarrh, or First Division of Bronchitis.

During the dry or inflammatory stage, the treatment should be for the purpose: First of abortion; second of hastening and promoting secretion.

Abortion to be successful must be attempted very early. In ordinary catarrh it may be affected with quinine and Dover's powder or other preparations of opium, given after a foot bath. The patient should be placed in bed, and kept covered, but should not be loaded with covering. The object is gentle perspiration.

This will be promoted by using a snuff composed of salicine one drachm, chlorate of potash one scruple, and pulverized gum acacia half an ounce. This may be drawn up into the nasal passages by snuffing, or be thrown up by an instrument.

If taken early the attack may be arrested. If, however, the opportunity of abortion is neglected, the next best thing to do is to hurry the natural method of cure by promoting free secretion. In addition to the abortive methods warm vapor may be inhaled, and attention should be paid to the digestive organs. Judicious stimulation may also assist.

A mixture of chloride of ammonium three drachms, chlorate of potash one or two drachms, cinnamon water six ounces, syr. senega and sweet spirits of nitre, each one ounce, with extract of licorice to disguise taste, may be of great benefit, when the throat is severely attacked. This may be given, tablespoonful to an adult, every half hour or every two hours, according to the results obtainable. Influenza or grippe may be broken up, if taken very early, by the following prescription: Half an ounce of chloride of ammonium, half an ounce

of nitrate of potash, half an ounce of senega root, and one ounce of licorice root; one pint of boiling water, infusion.

If the patient toasts his feet before a brisk fire, or places them in a hot *foot* bath, and takes of this infusion one tablespoonful every half hour during an afternoon and evening, and then retires to a comfortable bed, he may arise the next morning entirely free from the attack.

The early and efficient treatment of acute bronchial catarrh is but prudent forethought. It is true that an attack may run an even and uncomplicated course without medication, ending in perfect recovery, but there is always danger that the inflammation may extend to the fibrous sheath, which may be the beginning of serious complications, ending in fibrous phthisis.

Severe or inflammatory bronchitis is characterized by higher temperature, severer constitutional symptoms, and graver complications than the conditions of simple catarrh. The inflammation extends into the fibrous sheath, and frequently beyond it into the connective tissue of the peribronchic spaces. Peribronchitis, with inflammation of the fibrous sheath, has no regular course, but may continue for months or years, and then it is called chronic bronchitis. It does not extend through the whole of a bronchus, but is confined to points of limited extent. It results in stricture and corresponding dilatation, and is always complicated more or less with inter-pleural pathological results, adhesions, and thickened pleura. In post-mortem examinations the evidence of simple catarrhal bronchitis may entirely disappear or be so faint as to escape detection. But inflammation of the sheath

and cavities or tubercular nodules may be forme
the lung, which softening and opening into a b
chus may cause pneumorrhagia or fatal hæmopt
Or they may open into the pleural cavity, cau
hydro-pneumo-thorax.

Simple catarrhal bronchitis may thus end.

## ARTICLE XI.

### Chronic Pleurisy.

If we may include under this term all of the pathological causes and results of inter-pleural effusions of fluids and of exudations of plastic, fibroid, albuminoid, and other exudative matter, whether as the result of inflammation or of simple atony of tissues, then the subject is comprehensive and makes it necessary to glance hurriedly at the formative causes.

There may be three divisions of this subject. The acute inflammatory, the sub-acute inflammatory, and the passive or non-inflammatory.

Acute Inflammatory Pleurisy comes suddenly with a chill followed by violent pain and high temperature, and may end fatally at the onset, or favorably with effusion of serum into the pleural cavity.

Its formative history goes back but a short time, and generally where fluid is effused and is removed there follows speedy convalescence.

But causes may intervene to prevent or retard recovery. The fluid may become purulent, or a large amount of albuminoid and fibroid exudation may have taken place, and then we have chronic pleurisy with its complications and disabilities.

*Sub-Acute Pleurisy.*—Plastic exudation within the pleural cavity is one of the commonest pathological results of what we call a succession of colds and bronchial attacks. The physical signs of sub-crepitant râles, generally misinterpreted as being evidence of bronchitis, are really inter-pleural and denote plastic exudation and should be called plastic râles.

Usually the fresh exudation is rapidly re-absorbed,

but if the patient is reduced in vitality it may remain and become organized into adhesions or thickened pleura.

Wise management and medication may hasten and ensure its re-absorption when recent, and for this reason bronchial attacks should receive careful attention; for while many times they are but temporary indispositions, yet at others assistance is necessary, and the longer it is deferred the more difficult it becomes.

The sub-acute form of pleurisy occupies a place midway between the acute sthenic form and simple plastic exudation in which there is no rise of temperature nor pain, nor any of the accompaniments of inflammation; which is the third division of this subject.

*Plastic Exudation, Non-Inflammatory.*—The etiology of plastic exudation from mental or nervous depression may extend backwards for months or years, or it may result from intense mental worry and depression into acute plastic exudation.

Any cause which depresses the vital power and lessens the vitality of the blood may result in plastic exudation, the lax condition of the tissues favoring the transudation.

Worry, disappointment, despair, are the emotional factors. Atmospheric influences, of a depressing character, greatly add to the mental causes. It is characteristic of this disease that exudations occur periodically, which at first are like thin glue, almost as fluid as serum. But organization commences immediately. I have had the opportunity to observe a plastic hyperæmia of the lung in progressive pleuro-pneumonia in a cow. It was of only a few hours continuance, yet there were already signs of fluid plastic exudation within the pleural cavity, which could be heard as muffled, moistened respiration. At the same time a slight, tearing

sound occurred at intervals, as the ear passed over the surface, like tearing of wet paper.

The post-mortem which immediately followed the physical examination showed, as was diagnosticated, a thin fluid exudation covering the pleural surface. There were slight filaments of forming membrane branching in different directions from a central point. They were scattered here and there, and could be lifted upon the point of a knife. The movement of the lung in respiration parted these filaments and caused the slight tearing râles.

Organized plastic matter becomes adhesions when attached to both pulmonary inter-lobular surfaces, or to the pulmonary and costal pleura, or to the pericardial sac. If attached to one surface only, it becomes *thickened* pleura.

All of these forms of exudative pleurisy have similar inter-pleural pathological products, and permanently lower the vital power of the individual. They lessen the capacity for blood-aëration and consequently the amount of blood lessens and the patient loses weight and strength, and ability to assimilate food, and in this state slighter causes increase the pulmonary hyperæmia and new plastic matter is thrown out to increase and to extend the disability. The organized exudation which was caused by mental depression primarily, becomes itself a persistent physical factor of vital depression and results finally in progressive fibroid phthisis. Peri-bronchitis at the same time is also progressive as a part of the same pathological process.

The organized exudation continues to contract, obeying the natural law, and if it covers a large surface of the lung, it thereby shuts off the capillary circulation both of the pulmonary and of the nutrient arteries, which immediately subtend the pleural surface so

covered. At the same time the inflammatory products in the fibrous bronchial tube and the peri-bronchitis more directly obstruct the bronchial and pulmonary nutrient arteries.

This untoward state of things gives rise to many interesting phenomena not fully understood, except by those who search for primary causes and look beyond the immediately obvious for the essential causes of disease.

In this way not only does the contracting pseudo-membrane lessen the area for capillary distribution of pulmonic blood for aëration, but it also shuts off the circulation of the nutrient artery of the true respiratory system.

The nutrient artery is derived from the bronchial artery with additions from the mammary and the intercostal, but has this unique peculiarity that it has no *venæ comites* or returning veins.

The capillaries of this artery after performing their office of nutrition in the true respiratory system, pass their blood into radicles common to themselves, and to the capillaries of the pulmonary artery—the radicles of the pulmonary vein—which carry all the purified blood to the left heart for systemic circulation.

All varieties of chronic pleurisy have one common effect, that of interfering with the aëration, and circulation of the blood, and also lowering of the vital capacity of the patient. They differ in these particulars that, acute sthenic pleurisy when it becomes chronic, generally affects but one side, and may give rise to curvature of the spine, but is not so liable to end in pulmonary phthisis. The depressing causes which were mainly or wholly efficient in the second and third varieties in precipitating the primary attack have but little to do as causes in the first, but continue

to act as depressing factors in the second and third—more especially in the third—and it is in these two last that I am especially interested, for the knowledge of them comes to the physician as well as to the patient and friends as a surprise. Frequently in the subacute inflammatory variety the bronchial attack has been forgotten, and the attention is only drawn to the rational and physical signs of inter-pleural plastic results which are apt to be mistaken for "tuberculosis," especially if bronchorrhagia has taken place.

The malign effect upon all concerned of such a mistake is to prevent the use of effective means to prevent the phthisical result while it is yet remediable. For the tendency of the results of both the second and the third varieties, is to end in phthisis, either fibroid or tuberculated fibroid. I would make this distinction, that fibroid, which is frequently lingering, and is more amenable to treatment, is yet often fatal, but never becomes cavicular, except it first becomes tuberculated, that is, unless caseous degeneration takes place. Uncomplicated tubercular phthisis is a rare disease, and the few cases which I have seen, presented none of the physical signs which are depended upon in making a diagnosis of phthisis. For without adhesions of the lung to the chest-wall there is no telegraphy nor phonographic relations established by which centric changes may be comprehended.

The treatment of the first variety should be prompt at the outset, and if possible abortive. But if effusion of serum take place, the system should be allowed to rest for a week or more with palliative medication only, unless there is great suffering from dyspnœa. If that is the case it will be best to draw off a portion of the fluid at once. It is better not to interfere, however, unless the dyspnœa be great, as keeping the

pleuræ apart for a time prevents adhesions, and subsequent disability. If the fluid is not absorbed or lessened in a week or ten days it will be best to interfere and withdraw it—perhaps not all at once but gradually. Many times after a partial withdrawal with aspirator or trochar, the remainder will be speedily absorbed and healthful conditions will be resumed. When, unfortunately, adhesion and inter-pleural pathological products remain, nature may need assistance to remove them. Fresh air, out-door life, will do much, and may be sufficient. But if these fail, after a short trial, vigorous anti-plastic treatment should not too long be delayed.

The best medicinal treatment is the mercurial, in small doses in combination or otherwise, until slight constitutional effects are produced. Then changing to chloride of ammonium, or iodide of potash. Alternations of the mercurial and salt absolvents should be continued until the lungs are free in their movements. Outside medication should not be omitted. Spirits of turpentine is the best for recent exudation, then iodine, and lastly cantharides. Tonics should be given where indicated. The lungs should be systematically expanded by filling them constantly with air and holding the breath. The food should be nutritious and of easy digestion. Milk is the type, and it relieves the kidneys, also, in carrying out of the system tissue detritus.

# PART II.

## THE HEART.

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### INTRODUCTION.

THE first paper on cardiac murmurs was written for the purpose of bringing before the profession the value and certainty of the sign of Mitral Regurgitation as taught by the late Dr. Geo. P. Cammann. It was read before the New York County Medical Society, and afterwards was published in the *New York Medical Journal*, June, 1868.

Dr. Cammann had been in the habit of pointing out this sign as diagnostic of mitral deficiency before his class at the Demilt Dispensary. He also taught the unreliability of murmurs heard at or near the apex beat as an alleged sign of mitral regurgitation.

But all that he had written on the subject of cardiac murmurs was in the condensed summary which he gave me in manuscript (and another corrected copy found among his papers, and which was presented as an addendum to this paper, five years after his death).

I had several times made post-mortem examinations for Dr. Cammann which proved the existence of mitral incapacity in cases in which during life the characteristic sign in Cammann had been recognized.

This sign was a decided murmur heard to the left of the spinal column between the 7th and 8th vertebræ of the left side. I had also seen with Dr. Cammann cases of heart disease where this characteristic sign

was absent, but in which murmurs heard at the apex beat were present, in which there was no mitral incapacity found after death.

In order to place this matter in as clear a light as possible it was necessary to pass in review the accepted opinions of writers and teachers upon the physical signs of cardiac pathology and to compare them with the philosophy of heart sounds and murmurs.

At the very entrance into this subject I was met by the fact that much that had been written on cardiac signs was hypothetical. Some early distinguished teacher had evidently written of them in his study as it seemed to him that they should be, without comparing the signs of disease in the living with pathological evidences after death. These views, received with the stamp of authority of a great name, were added to by succeeding writers, resulting in a mass of misleading literature, difficult to unlearn, but which must be cleared away before the truth can be established.

The general disregard of writers on chest diseases of the laws of acoustics suggests their ignorance of them, which is probably true; for acoustics could hardly be considered an applied science until within these few years, since the introduction of the telephone into daily life. But the absurdity of writing on a scientific subject without applying to it the laws pertaining to that science should deter us from discarding a mass of mystifying hypotheses. Yet simple facts were observed and recorded and experiments were made which fix certain points beyond the domain of cavil, as was done in regard to the second sound of the heart being caused by the resiliency of the aorta forcing the blood back against the semilunar valves and closing them with a shock. This was determined by experiment as well as by observation; as

when the valves were hooked up or destroyed, the sound was absent. When the valve was merely injured, the second sound was altered in character or was supplanted by a diastolic murmur. This also enabled us to understand the philosophy of the rhythm of the heart, and of the intervals of silence. The systole of the heart having taken place, accompanied and characterized by the first sound, the heart rests, relaxes and passively fills slowly with blood. If there were no closure of aortic valves there would be but one prolonged interval of silence, until the heart should again commence to contract. But the second sound accompanying the forcible closure of the aortic valve divides the interval of silence into two of unequal times. So that we have established beyond controversy the facts of the first or short interval of silence, the second or short sound and the long interval of silence.

The first sound alone remains the subject of changing opinion, of doubt and false diagnosis.

If by applying acoustic law and the law of physics to the philosophy of the first sound, and the systole of the heart, we succeed in demonstrating their causes scientifically, we may remove this vexed subject from complex disputation.

The first sound occurs during the systole of the heart. It must be caused then by the contraction of the heart, as one of the factors. But the sound will not take place even if the heart contracts unless the heart is filled with blood. It has been demonstrated that the heart contracting while empty will be unaccompanied with sound.

Hence it is inferable that were the heart a closed cavity and were full of blood, the contraction would not cause the first sound, nor anything like it.

The blood must be forced out through an orifice to

cause any sound at all like the first sound. And with all these conditions perfect, and acting correctly, the sound caused will not be the natural first sound. There must be a mitral valve, thin and vibratory, held in its place by tendinous cords, and then only with all the other conditions acting perfectly and in harmony, will we have the first sound harmonious and natural.

The conditions for the production of the first sound are a perfect heart contracting upon and forcing out blood from its cavity into the aorta prepared for its active reception. The perfect heart must have all its interior appliances in a perfect state—mitral valve, chordæ tendineæ, muscular walls, columnæ carnæ, and musculi papillares. The contraction also must be regular and not spasmodic, and the blood must be of its proper consistency, or we will not have the harmonious and rhythmical first sound. It goes without saying that a sound-producing instrument of such delicate construction, and controlled by a sensitive nervous system often under the influence of external causes, must frequently be out of order, so as to be noted by the variations of the systolic sound without serious structural change invaliding its mechanical efficiency as a pump.

These functional murmurs come and go and have an extra-cardiac significance. Other murmurs of a more permanent character are caused by structural change or pathological results in the chest-wall or in the lungs, or by attachments of the pericardial sac and the pleuræ, but still are extra cardiac, and do not interfere essentially with the pump function of the heart. And there are structural changes in the heart itself which may weaken its muscular contraction, but still its normal function will remain, but it will be accompanied by alterations of the first sound characterizing the gravity

of the lesion without changing the rhythm. All of these murmurs are apex-beat murmurs and are intra-ventricular in cause and site.

But where there is obstruction to the flow of blood into the aorta, causing hypertrophy, or where there is incapacity of the aortic valve or the mitral valve, then the rhythm will be altered or lost, and the first sound essentially changed in character, and the proper relation of time of the first sound and first interval of silence, the second sound and the second interval of silence, will be altered, and is of far graver import to the life of the individual than murmurs, although they also have their value when rightly diagnosticated. But that is wherein the value of a scientific and consequently correct diagnosis has its highest appreciation to distinguish the functional and extra cardiac lesions from those invalidating the valves or weakening the heart's action: In one the heart may remain through life capable of successfully performing its office; the other indicates the beginning of farther structural changes, ending either in sudden death or prolonged and agonizing suffering.

The aortic obstruction and aortic regurgitation areas are practically unvarying; Cammann's sign of mitral regurgitation is also stationary. The mitral obstruction or mitral non-regurgitant murmur areas may have great variation by dislocation of the apex beat, which is not fixed, except as a result of disease.

## ARTICLE I.

### Cardiac Murmurs.*

Before we can properly appreciate the significance of cardiac murmurs we must be able to demonstrate the natural sounds of the heart, or, by induction, to approach so *nearly* to demonstration that exact experiment will scarcely be necessary to make the truth more plain.

We propose, also, to consider the human chest as an acoustic instrument, a sound-bearing and multiplying chamber, as well as to dispose of all ephemeral murmurs, preliminary to entering into the discussion of the philosophy of diseased or structural murmurs.

The sounds of the heart are two, the first long and the second short; the periods of silence or rest are also two, the first short and the second long.

The first sound is long, commencing with a low moan, growing louder and rising in pitch as it approaches the ear; it ends *with* and is emphasized *by* the impulse-beat. Then follows the short period of silence, which is immediately interrupted by the second sound, which is also very short and flat in character; and, lastly, comes the long period of silence.

The first sound, and the second period of silence, in a healthy heart beating deliberately, take up much the greater part of the time in the round necessary to complete the act of impelling the blood into the arterial system.

Physiologists are not agreed as to the mechanism of

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* Read before the New York County Medical Society, April, 1868.

the first sound. On the short period of silence, the second sound, and the long period of silence, there is no controversy. It has been proved by direct experiment that the second sound is caused by the sudden closing of the semilunar valves by the return shock of blood. A little hook passed into the aorta may hold up a curtain of the valve, when the sound will be absent. It is also absent when disease has destroyed the valve. The short period of silence and the long period of silence are made long and short by the second sound dividing the period which elapses from the time when the heart ceases to contract till it commences again.

The heart has one period of action and one of repose. This, really, is all the heart has to do with it. The second sound is formed independently of the heart by the return flow of blood in the aorta against and closing the semilunar valve, dividing the period of the heart's rest into two unequal parts. I shall not attempt to controvert the theory of active dilatation of the heart. I only desire to keep the simple fact clear before the mind that the heart acts, and then rests, agreeing with the law that muscular action or contraction is always followed by relaxation, and it would be singular if nature should make an exception in so important a muscle as the heart.

### THE FIRST SOUND.

The mechanism of the first sound is still *sub-judice*. If the difficulties environing this subject were swept out of the way, and the cause of the first sound were made plain and convincing, it would lift the unsatisfactory points of cardiac murmurs from the obscurity in which they have so long been enveloped, and place them in a clear light.

The majority of writers on cardiac sounds give prominence to three different theories. First, that of the friction of the blood in its motion within the ventricle and in its passage into the aorta. Second, that of the muscular contraction of the heart itself causing sound. This theory is based on the discovery of Dr. Wollaston, published in the "Philosophical Transactions of Great Britain" in 1810, that muscular contractions cause sonorous vibrations. Third, that of the vibrations of the mitral valve caused by its closure and tension and the forcing and rushing blood.

There are other theories that scarcely need to be noticed, as they fail to satisfy any acoustic law.

Some, recognizing the possibility of each of the three causes mentioned producing sound, have believed, as the first sound is evidently composite, that it is the result of all three.

This was Dr. Cammann's opinion, and it has a greater weight of probability and more proof than either theory alone.

It is clear, however, that the cause of the first sound must be in full agreement with acoustic law. Let us see if these separate theories agree equally with the facts and the law, or if a combination of these theoretical causes can produce the first sound.

The friction of the blood, in its motion within the ventricle and in its passage into the aorta, we can imagine could produce sound; yet in a state of health, that friction must be of minimum amount, for nature does not create obstacles in her own way. But if sound from this cause could be heard at all, it would be entirely different from what we actually hear. Blood in motion in a tube or vessel of irregular calibre would produce a rushing tube-friction sound. It would not be vocal, nor musical, and would have no quality

like that of the first sound, and therefore must be excluded.

Contracting muscle undoubtedly produces sound, but it passes no sound vibrations into the air. In order to hear the vibrations of contracting muscle, it is necessary that a sonorous body should convey them to the ear.

The sound is of very low note, the lowest that can be made by a piano string, having about thirty-two vibrations in a second.

Dr. Wollaston called the sound a susurrus, that is, a muttering sound, and likened it to the sound of a carriage at night in a distant street driven rapidly over block pavement.

Any one can hear it by placing his thumbs in his ears and resting his elbows on a table, or by closing the teeth tightly together, when all is still at night, with the head resting on the pillow.

This theory was the first that was offered to explain the first sound. But it is unlike it, having but one low note, while the natural first sound runs from the lower to the higher in regular gradation.

The third theory advanced, the vibration of the tense mitral valve in the presence of rushing blood, has greater probability, for it is based on acoustic truth.

But the simple closure of the valve as an act does not cause the sound, nor any part of it; it merely prepares the way. The valve being made tense is fitted to receive and reproduce vibrations brought to it—as we shall explain presently—and of passing these sonorous vibrations into the air, so that they may be heard without placing the ear in contact with the vibrating body. And yet this, without a more active cause added, does not account for the first sound. The first sound commences with the low pitch of a muscular susurrus; it

is musical in character, which a fluid friction sound is not. Again, the tense mitral valve, resisting forcing, rushing blood, would not of itself originate sound of the character which we hear; another sound-producing element is necessary to account for the low note gradually running up to higher pitch, like the string of a musical instrument having its tension gradually increased by the tuner while it is vibrating.

Failing to be satisfied with either of the three reasons considered, or in their combination, because they do not acoustically demonstrate the first sound, let us examine the heart anew, and see if there belong to it any other sound-making apparatus that will fully explain, physically and acoustically, all the peculiarities of the first sound.

We naturally give our attention first to the interior of the ventricle, and we find there rough walls strengthened by fleshy columns, to which are attached tendinous strings running athwart the ventricular cavity to be attached to the mitral valve. Their object is to hold the valve from being forced from its integrity, and the contraction of the ventricular walls, with the columnæ carnæ and musculi papillares, are so beautifully contrived that exact coaptation is always perfectly maintained, so long as the valve is sufficient, no matter what functional disturbance or emotional excitement or other conditions may occur.

The valve is thin and strong, and when tense is capable of reproducing and multiplying vibrations conveyed to it of a loud and sonorous character, though not originating them. The union of the chordæ tendinæ with the valve is an apparatus quite competent to produce all the characteristics of the first sound and to demonstrate it acoustically.

The tendinous strings, stretched across the cavity of

the ventricle and rendered tense by muscular contraction, are the very type of a sound-producing instrument. The rushing of blood among these cords must cause vibration, which, being multiplied and reproduced in the tense mitral valve, are readily passed into the air and heard without the chest wall. It seems strange that auscultators should generally have overlooked the chordæ tendinæ as the main instrument in the production of the first sound. We might as well attempt to account for the sound of the violin without the strings as for the first sound without the chordæ tendinæ.

Let us return to the study of the beautiful mechanism of the first sound, and suppose the ventricle has been filled in the natural way—the relaxed muscular tissue of the heart has allowed the blood, welling up to the auricle, to flow freely into the ventricle through the open auriculo-ventricular opening, till it has floated the mitral valve up to its position, closing, but without force, the auriculo-ventricular opening, the heart remaining passive, being dilated by the flow of blood only. But in due course, the auricle also becoming filled is stimulated to contract, which it does, and sends a wave of impulsion into the already filled ventricle, which, on the principle of the hydrostatic press, produces equal pressure on every part of the ventricular wall, which the ventricle acknowledging as its proper stimulus, immediately contracts, *instantly* closing the mitral valve, making tense the chordæ tendinæ, and sending the blood in its arterial course. The motion of contraction passes from the auricle downwards, and runs along the ventricular wall, and through the columnæ carnæ, exactly adjusting the tension of the chordæ tendinæ, so that the mitral valve is kept in perfect coaptation, resisting the mighty force of the

contracting heart, not one drop of blood being regurgitated, but all is hurled onward in its course. The resilient aorta sends back the column of blood against the semilunar valve, closing it with a shock, and the heart, exhausted, as it were, by the tremendous effort, lies relaxed and resting, waiting to perform the next beat in the same way.

Now, let us consider the character of the sound caused by the heart's contraction. It commences in a low moan, rising in pitch, and approaching the ear as it progresses, and ends with the impulse beat.

The acoustic laws concerned in this sound are in beautiful harmony with the mechanism. At the commencement of the sound the ventricle is full of blood, and the contraction makes tense, valve, chordæ tendinæ, columnæ carnæ, and ventricular walls; the rushing blood has not yet attained its maximum velocity, and the upper chordæ, which are the more tense, vibrate with the motion of the blood slowly, and the valve reproduces and multiplies the vibrations, and the low drum-like note is the result. But as the tension of the chordæ increases emptying the ventricle, the sound agrees with the facts and the acoustic conditions, and becomes louder, nearer, and raised in pitch to the end.

No other theory but this accounts for the character and quality of the first sound, that harmonious note of nature, the song of health.

In our study of cardiac murmurs we will have frequent occasion to make reference to the "chordæ tendinæ and mitral valve theory" of the first sound, which is based on acoustic law, and which is as perfect a demonstration as we can have or expect to have.

### THE HUMAN CHEST AS AN ACOUSTIC INSTRUMENT.

The human chest is an admirable instrument for

multiplying and reproducing sound. It is in the form of a truncated cone. Behind, the spinal column and the firm articulation of the ribs make a basic sounding-board. In front, the sternum attached by flexible cartilages to the ribs, allowing of considerable motion, acts as a counter-sounding-board, which may be brought nearer or removed further, and adjusted to the exact position for producing just the amount or volume of sound required. Below, where expansion may be most required, we find its capacity greatest, while above, where form alone is necessary, it is almost immovable. Then, the diaphragm closing the lower part of the chest has great latitude of motion, and can increase or diminish the sound capacity of the chest at will. It is thin and tendinous, and may be fixed in tension high up in the chest, or low down, just as may be required for the purpose of forming, increasing, or diminishing sound.

Man has not invented and may not construct a musical instrument of such varied applicability and such marvellous power. Ventriloquism is but the ingenious use of this power, for all its remarkable sound deceptions depend upon the educated diaphragm, modifying the quality of the sound of the voice. Song and speech depend on the perfection of the human chest as an acoustic instrument for their power to enchant us with melody or to astonish us with the forcible expression of thought. The violin, the most perfect of human instruments, is formed on the model of the human chest—it has its two sounding-boards, one at the back and one in front, and it has sides and ribs. Yet it has no flexible cartilages or ribs; the anterior sounding-board cannot be brought nearer or removed further, and it has no self-adjusting diaphragm; and we may well deem it beyond the power of man to construct an instrument of equal capacity with the human chest out

of unsentient materials. The violin is but the analogy of the human chest. The vibrations of the vocal cords, or the strings of the violin, are reproduced and multiplied indefinitely in the sound chamber of the human chest or the violin; they would have no volume, no reverberation, no *timbre*, removed from the acoustic instrument. Let the string be attached to a non-sonorous body and it will vibrate as well, and the pitch will be according to the rapidity of the vibrations, but the sound will have no quality above that of a child's toy. The volume and quality of sound do not depend upon the vibrations of the string, but upon the reproducing and multiplying instrument to which it is attached. Let the instrument be ever so little injured in its acoustic conditions, the alteration in the volume and quality of sound will measure the injury. Place a non-vibrating body upon the violin, or pour sand or shot or water into it, and its power of reproducing and multiplying sound will be notably impaired, and the same is true of the human chest.

The lungs are constantly filled with air, dilating every air sac, which by active resistance and forcible contraction compresses the residual air, increasing its sonorous capacity; whilst the convective air-tubes convey the sounds, like speaking-tubes, in every direction. This completes the perfection of the human chest as a musical or acoustical instrument.

Emphysema, or consolidation of the lung from any cause, or an enlarged heart, or an aneurism, or a tumor, or pleural effusion, may impair the acoustic qualities of the chest; and consequently the study of this subject is one of great importance to the auscultator, and this is especially true in regard to cardiac murmurs.

In rapid rhythm of the heart's action a murmur may appear but feeble to the unpractised ear, and when the

heart becomes irregular and tumultous, it may become difficult even for the expert to read its entire significance, but should pneumonia with consolidation or pleural effusion occur, all the murmurs would be enfeebled or disappear altogether. I have known a loud double murmur denoting obstruction at the aortic orifice and incapacity of the aortic valve, to disappear to the attendant physician and to so diminish in intensity during an attack of pneumonia as to be scarcely heard by an expert, and, remembering this acoustic fact, above stated, I passed my ear to the back part of the chest, and found to be true, what I suspected, that consolidation had taken place. In this case the pulse, usually about fifty in a minute, was not increased above eighty, and was not diminished in force.

Pneumonia and pleuritis are not unfrequent complications of cardiac disease, and a cardiac murmur suddenly diminishing in intensity, or disappearing altogether, may direct attention to a pathological fact, and assist in making out a correct diagnosis.

The philosopy of this novel and interesting acoustic physical sign may be demonstrated by placing a watch or a small music-box within a sound chamber, as of a violin or violoncello, taking care that it shall not touch the walls of the chamber in any way which might convey direct vibrations, and then to listen with the ear or a stethoscope against the outside of the chamber. Notice the clearness and distinctness with which even the lower notes can be heard, and then, while still listening, let an assistant pour water or sand into the chamber, and then to notice again the gradually diminishing of the intensity of sounds until they grow very feeble, to disappear entirely, especially the lower notes.

With this brief consideration of these two preliminary subjects, some knowledge of which I deem abso-

lutely essential to a proper understanding of the diseased heart sounds, we may turn our attention to

### CARDIAC MURMURS.

CARDIAC murmurs may be divided into those which are signs of functional disturbance and those which denote structural disease of the heart.

Functional murmurs may be divided into three kinds, those depending upon anæmia, those depending upon plethora, and those depending upon disease in some other organ acting through sympathy.

The anæmic murmur is generally easy of diagnosis. The marked anæmic condition will direct the attention from the first. The murmur is loud and diffused, heard over the base of the heart, and is carried thence in every direction over the chest. It is increased by slight exertion, and has no point of particular intensity, except at the apex beat, which distinguishes it from a structural murmur. It is very noisy, and may mislead the inexperienced.

The plethoric murmur may be heard where there is a full habit with an excitable condition of the nervous system. It is most frequently heard in pregnancy, and may, sometimes, assist in making a diagnosis of that condition. The murmur is heard over the base of the heart, as are all functional murmurs, but is not loud like the anæmic murmur, neither is it heard over distant parts of the chest; it is heard alone in the region of the heart, and has a low, muffled character.

A sympathetic functional murmur has its cause in disease of some other organ, as the brain, stomach, or uterus, and is not necessarily accompanied with either anæmia or plethora, and is caused wholly by an excited state of the nervous system. Direct conduction of sound by adherent pericardial sac to lung and again to

chest wall or mediastinum is a frequent cause of false diagnosis of cardiac disease.

All functional murmurs are somewhat intermittent, and always pass away with the removal of the cause. They all have their site within the ventricle, an are owing, mainly, to irregular contraction of the columnæ carnæ, the musculi papillares and the ventricular wall, bringing the chordæ tendinæ into irregular tension, and causing discord in the natural first sound.

The varying conditions of the blood account for the differences in the character of the three varieties of functional murmurs. In anæmia the blood-vessels are not distended, the general acoustic qualities of the chest are increased, and the blood rushes along, carrying the murmur far into the blood-vessels, from which sonorous vibrations are passed into every part of the chest wall. In plethoric murmur the acoustic conditions are decreased, and the murmur is carried but a short distance from the heart; and in functional murmurs from extrinsic disease, the conditions of the chest remaining natural, the murmur will not be so loud, nor will it be carried so far as in anæmia, and yet it will be further than in plethora.

There are some functional murmurs that deserve particular attention. One is the systolic murmur heard in inflammatory rheumatism. It may cause needless alarm to those not fully apprehending its meaning, yet it is a warning to the intelligent physician that will direct his watchful attention to the heart. It is an intra-ventricular murmur, and we are enabled by the rules we have laid down to diagnosticate it differentially from an organic murmur. It is of harsh character, heard over the base of the heart, but not with maximum intensity at the apex beat, nor is it heard with particular emphasis at the aortic orifice, or

at the place where the aorta emerges beneath the sternum, nor under the clavicle. It is heard in the direction of the current of blood, but the sound is of a diffused character like other functional murmurs. Its character sometimes runs quickly into one denoting deposits of lymph upon the valves. If the murmur becomes suddenly distinct over the aortic valve, and is heard emphatically at, or near, the cartilage of the fourth rib of the right side and under the right clavicle, and on either side of the spine from the third to the sixth vertebræ behind, we know deposit has taken place at the aortic orifice; or if the murmur assumes maximum intensity at the apex beat and is of rasping character, we know that there has been deposit upon the mitral valve. This murmur has given rise to the opinion that acute articular rheumatism always produces some damage to the heart, which is not quite correct, for the murmur frequently subsides and passes away with the rheumatism, proving that it was only a functional murmur. It affords a valuable index to the treatment best calculated to prevent damage to the heart. Its cause is probably due to spasmodic contraction of the muscles of the heart from nervous excitability of the endocardium, due to the irritating quality of the blood.*

Adhesions of the lungs to the chest wall, to the mediastinum, and more especially to the pericardial pleura, as well as pericardial adhesions to the heart, also produce murmurs, and the murmurs continue as long as the adhesions may influence the symmetrical contraction of the ventricular wall. These murmurs are apt to mislead the practitioner into making a false

* There may also have occurred a removable deposit upon the mitral valve, which disappears with the inflammation.

diagnosis, but they have no important significance, for when the adhesions lengthen sufficiently the murmurs will disappear.

There is also a functional murmur, associated with chorea, which has been considered as the result of cardiac disease. Cardiac disease may be complicated with chorea, which is exceptional; the murmur of chorea is ephemeral, like all functional murmurs, and disappears with the cause. There is a peculiarity about the murmur of chorea that has given rise to the belief with some that it is caused by mitral regurgitation, because this murmur is emphasized at the apex beat. In the proper place we will endeavor to show that the apex-beat murmur is never a sign of mitral regurgitation. But it is a sign, if that were necessary in this disease, of unusual and violently irregular contraction of muscular tissue of the heart, that the chordæ tendinæ are so irregularly and so forcibly brought into tension that the vibrating murmur is conveyed in the muscular tissue of the heart to the chest wall, and of course will be emphasized at the apex beat. Regurgitation through the mitral valve never takes place except from insufficiency.

## CARDIAC MURMURS—ORGANIC.

The left side, or the left heart, being mostly in front and near the chest-wall and accessible to the ear, will be considered when we speak of cardiac murmurs. This is eminently proper, as the left heart performs the important office of impelling the blood into the system, has much greater muscular development, and is much more liable to organic disease than the right heart, and as they act in perfect synchronism in health, what is said of the left will be true of the right, with such exceptions as will be noted subsequently. The left heart,

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CARDIAC MURMURS.

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like the right, has an auricle and a ventricle, two valves, the aortic semilunar and the mitral auriculo-ventricular valve, and each valve may have two murmurs, the dual character of the heart always being maintained. The aortic valve may have two murmurs, the aortic obstructive systolic and the aortic regurgitant diastolic.

The aortic systolic obstructive murmur is caused by some impediment to the flow of blood at the aortic orifice, which may be deposits of lymph, or warts, or excrescences, or it may be what is called ossification—calcareous deposits at the aortic orifice or in the curtains of the valve. It must be something that will throw the current of blood into unusual vibration, and must agree acoustically with the physical facts. The murmur, from the manner of its formation, must have certain definite characteristics that will distinguish it from other murmurs; it must agree with the mechanism of its cause. We must insist upon this fundamental truth in regard to all the murmurs of the heart; the character of the murmur is an indication of the cause. In health the blood flows through the aortic orifice without murmur, the sounds of the heart are heard, if the ear be placed over the aortic valve, but nothing else. But let a deposit of lymph take place upon the valve, and notice of the fact will immediately be given by the murmur. What will be the character of that murmur? This we are able to demonstrate: fluid forced through a tube of equal calibre will cause no murmur, but if obstruction at a certain point be caused by pressure upon the tube, or otherwise, a murmur will be the immediate result. The character and quality of this murmur must be, from the identity of the cause, the same as is heard when there is obstruction at the aortic orifice; it will be a fluid friction sound, and have a rushing character. When hyper-

trophy has taken place, the murmur will be altererd or disguised by a vocal element of sound, which will be more particularly described when we come to speak of the mitral non-regurgitant murmur. The normal character of this murmur is only heard for a short time, for as soon as hypertrophy of the ventricle takes place as a result of the obstruction, the murmur heard will be of a composite character, for the mitral non-regurgitant murmur will be a part of the sound. It is well to keep this distinction before the mind, for the importance of the damage done is not measured by the noisy element of the intra-ventricular murmur, but rather by the character of the murmur formed at the aortic orifice. If the obstruction be but little, the murmur will be short in duration, not of high pitch, and will be heard at the same moment with the first sound, and will be of the character of fluid friction. If the obstruction be considerable, the murmur will be prolonged and of higher pitch, and will be more easily recognized by its dissimilarity from the natural first sound, especially when discord has been introduced by hypertrophy or by diseased mitral valve. This murmur may be heard best at certain points where the column of blood approaches the chest-wall.

Where the aorta emerges from under the sternum on the right side, near or above the cartilage of the fourth rib, will be one of these points; under the clavicle will be another; and posteriorly on either side of the spine from the third to the fifth vertebra, and on the right side running down the scapula to its lower angle are diagnostic points where we may search for this murmur when we have reason to fear the cause is established. It is rarely heard uncomplicated with other murmurs; but by experience the ear learns to

discriminate and to judge of the amount of obstruction and the probable damage. The murmurs are heard in the chest wall whenever the column of vibrating blood comes near enough to the chest wall to pass its sonorous vibrations into it.

### AORTIC REGURGITANT MURMUR.

The aortic diastolic regurgitant murmur is the second murmur heard in connection with the aortic valve. It is heard during the long period of silence, and with or immediately after the closure of the semilunar valve, and is caused by its insufficiency. As the result of disease or by violence, an opening is formed in the valve which allows a stream of blood to be thrown back into the ventricle. This murmur is uncomplicated, for the intra-ventricular murmurs are not heard during the diastole. It has only one quality, that of blood friction, and will be long or short, of raised or comparatively low pitch, according to the size or shape of the orifice allowing the regurgitation.

This sound may be accurately represented by forcing fluid through a syringe, and by altering the aperture of the nozzle imitate the characteristics of the aortic regurgitant murmur.

This murmur may be heard, and is most generally heard, about half an inch to an inch from the aortic valve, in a direction toward the apex beat. Sometimes it is heard as far as to the apex beat, and sometimes it is only heard through the sternum, and some distance from the aortic orifice.

The reasons for these variations depend upon the direction given to the stream of regurgitated blood and the proximity of the heart to the chest wall.

This murmur generally appears in the order of succession. If, during an attack of rheumatism, a deposit

of lymph occurs on the aortic valve, the murmur giving notice of the fact will be the obstructive murmur; the regurgitant murmur will not be heard at first, nor till some time after the plastic deposit following the law governing these deposits commences to contract, and then when the curtains of the valve can no more be brought into coaptation, regurgitation will ensue, and the diastolic murmur will be the sign. Or the cause may be warts or vegetations, or the deposit of calcareous matter, or it may be the result of violence, in which case it would not be preceded by the obstructive murmur. This murmur is sometimes difficult to hear. The gentle rush of blood, when the heart's action is irregular and tumultuous, requires an acute ear to catch the sound. Frequently the altered second sound gives warning that insufficiency of the valve is about to take place. This alteration will be, that while the second sound is more forcible than natural, it begins to lose in clearness, and has a muffled character.

### THE MITRAL VALVE.

The murmurs connected with the mitral valve are two; the mitral regurgitant and the mitral non-regurgitant or the intra-ventricular. They are both systolic murmurs. One has its diagnostic seat in the posterior chest wall, and the other in the anterior.

### THE MITRAL REGURGITANT SYSTOLIC MURMUR.

In studying this murmur we must first endeavor clearly to understand the cause; for the murmur when it is heard, to be truthfully explained, must agree not only with the physical conditions of the cause, but with acoustic law. The cause is simply insufficiency of the mitral valve. From disease or from

violence, or congenital malformation, an opening has been made or left in the valve, and when contraction of the ventricle takes place, and the valve is made tense by the forcing of the blood and the restraint of the chordæ tendinæ, a stream of blood will be violently rushed through the opening. This will cause a murmur the character of which will be determined by the size and form of the aperture.

It will be a blood friction murmur complicated with sonorous vibrations of the chordæ tendinæ of the mitral valve, and will be heard during the systole in the posterior chest wall.

Regurgitation through the mitral valve may be from congenital malformation, but it generally takes place after the valve has been damaged by disease.

The valve may be ruptured by violence, but this is a very unusual accident. Or, dilatation of the auriculo-ventricular orifice from degeneration of muscle may incapacitate the valve, but as a rule the murmur appears some time after a deposit of lymph has taken place, or from calcareous deposits.

The character of the murmur is evidence of the condition of the valve.

If the murmur be harsh and rasping as well as having the blood-friction, rushing character, we are safe in judging that the valve has lost its acoustic quality of reproducing sound; that it is damaged by hardened deposits of lymph or by calcareous deposits. But the fact should be severely questioned before it is admitted, for it is certain that inter-pleural adhesions attached to the pericardial sac restraining the heart's movement may give rise to a systolic murmur heard at the apex beat or in front, *all but* indistinguishable from that of a damaged but non-regurgitant mitral valve.

The murmur heard in front at the apex beat, may

give notice of these deposits upon the mitral valve and of their character. This harsh, rasping murmur is called the mitral regurgitant murmur by writers generally; but it is never a sign of regurgitation, but of deposits upon the valve, and its presence will give notice that regurgitation may take place, if it have not already.

Where shall we seek for the true regurgitant murmur? In the first place we must ascertain the direction of the regurgitated stream, for the sound vibrations are carried along with it and proceed in the direction in which it is sent.

If the stream strikes upon a substance capable of transmitting vibrations, they may be heard in the chest wall. If we suspect insufficiency of the mitral valve, it will become a certainty beyond cavil, if we hear a blood-friction sound between the seventh and eighth vertebræ close to their spines. It satisfies the ear that the cause is found, for it rushes into the ear, as it were, and has the same character as the aortic-regurgitant murmur, modified by the mitral valve; yet its characteristics will be recognized even in the presence of other murmurs.

Its maximum intensity is only heard between the seventh and eighth vertebræ, and there the character of the sound is diagnostic. It must not be confounded with the mitral non-regurgitant murmur which may sometimes be heard at the lower angle of the scapula, where also the aortic regurgitant is occasionally heard. The murmur may be heard from the lower border of the fifth to the upper border of the eighth vertebræ. but the characteristic murmur which renders the diagnosis certain is only heard between the seventh and eighth vertebræ; and unless heard here distinctly, regurgitation will not be proven, notwithstanding the

presence of other physical signs and rational symptoms, which are given by writers as signs of mitral regurgitation.

The anatomical reasons why the diagnostic regurgitant murmur should be heard between the seventh and eighth vertebræ are, to my mind, convincing.

When Dr. H. M. Sprague, U. S. A., was a member of the Examining Board in this city, in 1864 and '65, I requested him to demonstrate upon the cadaver the anatomical relations of the mitral valve and the left auricle with the organs between the auricle and the vertebræ, which he did a number of times, and gave me the following explanation:

"The left auriculo-ventricular opening lies over the seventh inter-vertebral space, the left auricle lying over the seventh vertebræ, having the œsophagus on the left and the aorta on the right, in immediate relation behind. The œsophagus overlaps the aorta somewhat in this region."

This is sufficient anatomical proof. The mitral valve lies over the seventh inter vertebral cartilage, and a regurgitant stream of blood would be thrown directly toward this cartilage, and the sound vibrations would be continued through the œsophagus, aorta, and cartilage to the ear. The mitral valve is near enough to allow vibrations to pass into the seventh vertebræ during regurgitation, and also the auricle lying upon it would pass vibrations into it. The pulmonary vein passing up over the sixth vertebræ would pass vibrations through it to its upper border.

All this will agree with Dr. Cammann's description, that the murmur may be heard from the lower border of the fifth to the upper border of the eighth,

with maximum intensity and characteristic quality between the seventh and eighth only.

Bellingham and others describe the murmur heard in front as diagnostic of mitral regurgitation, and succeeding writers and lecturers have taught the same doctrine till it has come to be the settled view of the profession. Yet I may run the risk of being called a "setter-forth of new doctrines," by attempting to prove Dr. Cammann's opinion to be correct and the generally received opinion to be in error.

In the *British and Foreign Medico-Chirurgical Review* of July, 1861, there is an article by J. S. Bristowe, M.D., Lond., F.R.C.P., Physician to St. Thomas's Hospital, on mitral regurgitation arising independently of organic disease of the valve.

Dr. Bristowe says that he had conducted the post-mortem examinations of medical cases at St. Thomas's Hospital for more than ten years. He says, "It by no means infrequently fell to my lot to inspect cases of reputed mitral disease, in which all the secondary effects of that lesion—pulmonary apoplexy, anasarca, nutmeg liver—were indisputably present, but in which the heart was found to present but little departure from the healthy state, and in which all the valvular structures appeared to be perfectly sound and competent. I have felt convinced, for some years past, that these cases were neither exceptional nor rare." Again he says, before detailing his six cases, "My first object will be to prove the fact of regurgitation through the left auriculo-ventricular orifice in certain cases in which the mitral valve is found to exhibit a perfectly healthy appearance, and to establish the frequency of its occurrence, by detailing such well-marked examples of the phenomenon in question as have occurred in the hospital during the four years above

specified." Dr. Bristowe's cases are related with minuteness, and give not only the signs observed during life, but also the post-mortem appearances.

After detailing his cases he says, "I have remarked that it may be regarded as an axiom, that the existence of a systolic murmur at the apex beat of the heart is a sure indication of incompetence of one or other of the auriculo-ventricular valves, and that so rarely is this phenomenon manifested in connection with the right side of the organ, that it might almost, for practical purposes, be accepted as the proof of mitral incompetence alone. This statement merely expresses the current doctrine of the day, a doctrine which no one will call in question, and one, indeed, which cannot be controverted without entirely upsetting the present well-established principles of cardiac pathology."

Dr. Bristowe states fairly the prevalent doctrines of the day, and yet his cases prove, if they prove anything, that that doctrine is an error; and that the apex-beat murmur is not a sign of mitral regurgitation.

The frequent exceptions which Dr. Bristowe mentions, where the apex-beat murmur failed to be a sign of diseased mitral valve, agrees with the experience of others, and completely invalidates its diagnostic value.

An apex-beat murmur is frequently but not always associated with regurgitation, and the regurgitation may take place with no murmur heard in front, and the apex-beat murmur is frequently present when there is no incompetency, and sometimes when there is no disease even of the valve. It would be interesting to know if in any of Dr. Bristowe's cases the regurgitant murmur could have been heard behind in its

proper place. In one of the cases detailed it is possible that the dilatation of the auriculo-ventricular orifice was sufficient to allow regurgitation; but it seems to me there could not have been in the five others. As laid down in books and taught didactically, there are a great many more cases of mitral regurgitation than aortic regurgitation, but the sign depended upon is fallacious. When we come to scrutinize these cases and apply the proper test we find them diminish to a small number, much less than the average number of aortic regurgitations. The average number of regurgitations through the tricuspid valve is still less.

Dr. Bristowe also refers to Mr. Wilkinson King's well-known paper, "On the Safety-Valve Function in the Right Ventricle of the Heart." He there attributes the regurgitation which, as a normal process, takes place occasionally through the tricuspid aperture, to temporary over-distension of the thin and yielding ventricular walls, and consequent displacement and insufficient length of the musculi papillares and chordæ tendinæ.

Dr. Bristowe, then, accepting Mr. King's theory as satisfactory in regard to the tricuspid valve, reasons that in dilatation of the left ventricle it would be assimilated in character to the right, and then the regurgitation might take place through the mitral valve But is it ever true with either the tricuspid or mitral valve that regurgitation takes place as a safety-valve function? I shall be slow to believe it. Are these not theories which are made necessary to explain the inconsistency of the apex-beat murmur as a sign of regurgitation? It seems to me Dr. Bristowe's article proves the necessity of reviewing "the current doctrine of the day" that the apex-beat murmur is a sign of regurgitation through either the tricuspid or mitral

valves; and I present Dr. Cammann's sign of a characteristic murmur heard between the seventh and eighth vertebræ as the only sign that really proves mitral regurgitation. This sign is infallible when clearly made out. It is possible that regurgitation may take place, and this sign be unheard; but if so, the fact is exceptional. I have never known a case.

The frequency of regurgitation through the different valves is the reverse of what has usually been taught. The possibility or probability of sudden death is a subject of alarming interest to the patient or to his friends. And for that reason aortic regurgitation has been looked upon as a fearful omen. Yet it is within the experience of every physician who has seen much practice, that incapacity of the aortic valve is not incompatible with a long life. If we reject the apex-beat murmur, and confine our diagnosis of mitral regurgitation to Dr. Cammann's sign of a characteristic murmur between the seventh and eighth vertebræ, agreeing with mitral insufficiency as shown by post-mortem examinations, the relative frequency of these regurgitations will be changed, and the aortic regurgitation will be first in the order of frequency, the mitral next, and probably the tricuspid last of all. I say *probably*, for I cannot point you to any certain, invariable sign of tricuspid regurgitation. Perhaps this alarming sign has been wisely hidden from us.

The following statement is probably correct. Incapacity of the aortic valve is of the greatest frequency, next of the mitral, third of the tricuspid, and of the pulmonary semilunar valves least of all, if at all. I do not know of a single well-authenticated case of insufficiency of the pulmonary valve existing for any length of time during life.

The origin of the manifestations of fœtal life are first

noticed in structural formation of the right auricle, and there also is noticed the last act of expiring functional life. It seems proper, then, that we should locate the point of greatest danger of sudden death in the right auricle, and that serious damage done to the tricuspid valve, involving its integrity, should be attended with great danger. The auriculo-ventriculo-septum of each side is probably the most sensitive part of the heart to morbid influences. Myocarditis, or other cause of softening of the heart muscles, may give rise to sudden death without valvular disease. With our present knowledge, it seems marvellous that the heart should go for so long a time under disability, and then, without any new condition being set up, suddenly to sulk and stop; and yet it is no more surprising than that it ever began to beat, or that it continues for years when once begun.

The danger of the heart's suddenly stopping is probably greater when there is extensive disease invading both hearts. But if influences received through the great organic nerve hurry the heart's action, while influences received through the pneumogastric slow it, the sudden stopping may be but a freak of nerve influence.

### THE MITRAL NON-REGURGITANT.

Those who have followed us in our study of the mechanism of the first sound will readily comprehend what we have to say on this subject, in a few words. We have described the first sound as being the result of blood rushing through and among the tense cordæ tendinæ of a contracting heart and of course throwing them into sonorous vibrations, which being reproduced in the tense mitral valve

cause a sound of a certain character. This sound is caused by a natural musical instrument, the heart, and like a perfect artificial musical instrument, discord is proof of derangement either functional or organic. I shall include in my description of the mitral non-regurgitant murmurs, all the murmurs having a cause in the mitral valve or chordæ tendinæ, whether functional or organic, whether owing to irregular contraction of the walls of the heart or columnæ carnæ, as in functional murmurs which disappear when the nervous system returns to a state of quiet health, or to organic change in the form of the heart or its muscular attachments, or to damage done by deposits on the mitral valve or the chordæ tendinæ. The murmur is always loud and noisy, and has infinite variety. It may be of no alarming import, or it may be an indication of serious damage done to the mitral valve. It may be a soft, blowing sound, diffused all over the chest, and yet seeming to follow the course of the blood-stream sent from the heart, or it may be louder, of a bellows character, heard with greatest intensity over the base of the heart, and extending but little into the column of flowing blood, and then it tells of hypertrophied ventricular walls. And if a murmur is heard in addition to this at the apex beat, loud and harsh, of varied pitch, rasping, sawing, blubbering, flapping, it is a sign that with the hypertrophy there is extensive damage done to the mitral valve. This murmur has its seat over the base of the heart, and at the apex beat, and may run round under the axilla and appear at the lower angle of the scapula behind, on the left side, or it may pass from the apex beat toward the sternum, just as the sound may be sent into the rib by the motion of the heart as it strikes the chest-wall. It adds something to the character of the aor-

tic obstructive murmur and to the mitral regurgitant, as heard between the seventh and eighth vertebræ behind. It attracts the attention of the beginner, for it is easily heard, and it frequently misleads the practitioner as to the gravity of the disease. It has been, in some of its varieties, considered a diagnostic sign of mitral regurgitation, and some varieties of it have been called by eminent auscultators a "presystolic murmur," or an "auricular systolic murmur," or a "mitral direct murmur."

Grisolle described what he called a presystolic murmur. Dr. Gairdner, of Edinburgh, describes the same murmur, and calls it an auricular systolic murmur; and our own eminent auscultator, Dr. Flint, calls it the mitral direct.

If we allow the cause to be as is described, the name auricular systolic would be most appropriate.

It is claimed to be heard just before the ventricular systole has commenced, and to be caused by the contraction of the auricle forcing the blood into the ventricle through a diseased and contracted auriculo-ventricular orifice, sometimes appearing like a buttonhole slit. The argument is, the murmur is heard, and the obstruction exists, therefore the forcible passage of blood through the narrowed orifice causes the murmur. We will endeavor to prove that the murmur is not caused by forcible passage of blood through the narrowed orifice, and secondly to account for the murmur in a more satisfactory manner.

The walls of the auricle are thin and its power is but feeble. It may be doubted that the auricle has sufficient power to force a stream of blood into the empty ventricle, so as to cause a murmur that would be audible at the apex beat. And it is incredible, for it is impossible that such a murmur could be formed when

the ventricle is full of blood. When we recollect that the murmur of regurgitation through the aortic valve is but feeble, and scarcely heard, notwithstanding the great force of the return blood by which it is made, or the true mitral-regurgitant murmur heard in its diagnosta place behind, which is not loud, yet easily observed, which is formed with all the force of the powerfully contracting ventricle; we cannot conceive that so feeble a cause, so far removed from the ear, can make so loud and harsh a murmur. Then, too, it must be remembered that the auricle and its appendix are rather a receptacle than a motive power. The auricle is not a shut sack, and it has no valve to prevent regurgitation towards the lungs; and that a bending or folding upon itself would not be sufficient to prevent the blood being sent back with damaging effect. Again, the murmur as heard is of considerable length in duration, while the time of the auricular contraction is exceedingly short.

Harvey, as well as other observers, since describes the motion of the auricle in contraction as beginning suddenly; a wave-like motion which passes immediately downward into the ventricle, instantly closing the mitral valve by contraction of the ventricle and sending the blood into the aorta. The murmur heard cannot be formed by the auricular systole, for there is no agreement in time.

And again, the murmur heard is entirely different from that of blood being rushed through an aperture, which would be like the sound of fluid being forced through the nozzle of a syringe into water, and would necessarily have a great degree of uniformity; while the sound actually heard is infinitely varied in quality, tone, and pitch.

And lastly, in disease of the mitral valve, intermission of the ventricular systole is a frequent occurrence, but not so with the auricular; that is not intermitted; and yet I have never heard, nor heard of, an auricular systolic murmur during a ventricular intermission. I have listened carefully to a heart with extensively diseased mitral valve, where the ventricular intermission was sixteen seconds in time, and during that intermission there was silence. Are not these facts satisfactory evidence that this murmur is not caused by the auricular systole?

How, then, is this murmur formed? If we refer back to the argument of the cause of the first sound, it will give us the key. These murmurs are mostly heard when the mitral valve is much diseased, of which they are a sign. The thickening and irregularity of the mitral valve, with the irregularly hypertrophied ventricular walls and columnæ carnæ, are the physical causes of the murmur. These will produce in contraction irregular tension of the chordæ tendinæ, and especially of those in the upper part of the ventricle. Some of these cords may have slight tension or none at all and vibrate slowly, producing a blubbering murmur; while others, at the same time, may be under great tension and give a harsh, rasping murmur of high pitch. Some from the altered form of the heart may be brought suddenly into tension with a snap as described by Dr. Ormerod. They are all formed at the commencement of the ventricular systole, as is proved by the preponderance of the mitral-valve element in their composition, and are only varieties of the non-regurgitant murmur, having their origin in the vibrations of the chordæ tendinæ reproduced in the tense-mitral

valve, and within the time of the first sound or ventricular systole.*

The murmurs connected with the right side of the heart are few, and all belong to the tricuspid valve. As before stated, the pulmonary semilunar valve is not liable to disease. The tricuspid is liable to the same damage from deposits, etc., as the mitral, but much less frequently. Sometimes in deformity of the chest from angular curvature of the spine, the heart may be so dislocated that the right heart might be brought near the chest wall, when its sounds may be studied in the same manner as we ordinarily study those of the left heart.

A tricuspid intra-ventricular murmur is not remarkably infrequent, but is much less frequent than the mitral. It is heard, ordinarily, at the lower part of the sternum, or by the left side of it, over the costal cartilages; or it may be heard at the upper part of the sternum, running out under the left clavicle. It has the same character as the mitral non-regurgitant; and though more distant from the ear and less sonorous, is evidently formed in the same way. The right ventricle is liable to hypertrophy from pulmonary obstruction, and this will produce the murmur described. This valve may also be damaged by deposits, etc., as the mitral is, and the diagnosis will be in the character of the murmur, heard over the cartilages by the left side of the xiphoid cartilage. I know of no certain sign of tricuspid regurgitation. The right auricle has its natural bed in a hollowing out, as it were, of the right lung in its middle part, and should there be

* Prof. Donaldson adopts this view in a paper entitled, "Significance of the Præsystolic Murmurs.— F. Donaldson, M.D., 1874."

a stream of blood regurgitated through the right auriculo-ventricular opening it would impinge upon the side of the auricle, and the murmur would be lost in its diffusion in the lung, and would not be brought to the chest wall, unless by consolidated lung. I have never heard it, and do not know that it has ever been verified.*

In a monograph on the "right side of the heart," by Thomas Mee Daldy, M.D., late President of the Hunterian Society, London, there is a condition pointed out which Dr. Daldy calls "a distensible right auricle." It is not accompanied with a murmur, but it causes the heart's sounds to be heard distinctly to the right of the sternum at the upper part and out under the clavicle, and there is dullness under percussion to the right of the sternum in the region of the auricle. This distensible condition is apt to be overlooked in post-mortem examination, for the auricle is not apparently diseased. But the fact may be demonstrated by filling the auricle with water, and by inspecting its bed in the lung, which will be found larger than usual.

Dr. Daldy says this condition is sometimes inherited, and is connected with dyspepsia. It is the cause of certain forms of asthma or apnœa, and of frequent congestive head-aches, which sometimes end in insanity.

I think I have verified the physical conditions described by Dr. Daldy in one or two instances.

In the foregoing paper I have endeavored to be practical, without claiming to be very original, and to give

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* I have since heard it in consolidated right lung.

my own experience as corroborative of that of the late Dr. Cammann.*

* *On Cardiac Murmurs.* By the late DR. CAMMANN, New York City.

[The following brief article, which has never before been published, although read before the New York Academy of Medicine, was found among Dr. Cammann's papers subsequent to his death. It is of importance in connection with Dr. Leaming's paper on the same subject, which is given above.—ED. NEW YORK MEDICAL JOURNAL.]

AORTIC OBSTRUCTIVE SYSTOLIC.

WHEN it reaches the apex it is with diminished intensity. When heard behind, it is most distinct at the left of the third and fourth vertebræ, close to their spines, and frequently extends downward along the spine in the course of the aorta, but with diminished intensity.

Although the heart extends only as high as the fifth vertebra, the murmur is heard above that point, because here the aorta approaches the surface.

AORTIC REGURGITANT DIASTOLIC.

Intensity from valve to right of apex may or may not increase downward, depending on proximity of the heart to the parietes, position of lungs, etc.; it may decrease downward, however, from emphysema, supine, recumbency, etc.; it may, perchance, be loudest at the apex, but depending on the proximity of the heart to the parietes, position of parts, condition of mitral valve, etc. Generally it is not heard behind, but it *may* be, towards the inner side of the lower angle of the scapula, in thin subjects especially, in the same place where is heard the mitral non-regurgitant murmur; this mitral non regurgitant being the mitral regurgitant of Bellingham and others. It is sometimes conveyed to the left axilla. The patient when recumbent may sometimes hear it himself.

MITRAL SYSTOLIC REGURGITANT.

To indicate regurgitation the murmur must be heard between the lower border of the fifth and the upper border of the eighth vertebræ, at the left of the spine, provided that the transmission of the sound be not interfered with by thickness of integuments or other condition of parts. When not heard in this place, but in the "left axilla and region of left scapula," regurgitation is

not indicated, or, in other words, it is a non-regurgitant murmur, contrary to the teaching of Bellingham and others. If there be a systolic murmur with a *maximum* of intensity between the seventh and eighth vertebræ at the left of the spine, it indicates regurgitation.

An aneurismal murmur, however, may be heard within the said limits, but it follows the aorta downward, gradually decreasing in intensity without the *abrupt* termination of the regurgitant murmur. We occasionally meet with mitral regurgitant murmur posteriorly yet absent anteriorly.

The following complication may exist, namely: aortic obstructive sytolic, with aortic regurgitant diastolic extending *to* the apex, with mitral regurgitant behind without a corresponding murmur in front. All of these murmurs are not unfrequently heard to the right of the apex, and even over the whole chest.

A mitral diastolic murmur we have not heard. If it be ever present, as stated by distinguished auscultators, it must depend upon physical conditions, external to the heart. Pleuritic effusions or the like in certain positions, by pressing suddenly and strongly upon the left auricle, may possibly force the blood with such rapidity through an obstructed auriculo-ventricular orifice, as to cause an abnormal sound.

Some auscultators, however, deny the possibility of the occurrence of this murmur under any contingency whatever.

*Addendum.*—The mitral-regurgitant murmur behind may disappear, from such a change in the structural condition of the deceased valve, or from such contraction of the auriculo-ventricular opening, as will allow the valve to close during the systole; there being, in this case, actually an increase of the mechanical obstruction.

## ARTICLE II.

### Significance of Disturbed Action and Functional Murmurs of the Heart.*

In April, 1868, I had the honor of reading a paper on "Cardiac Murmurs" before the New York County Medical Society, in which my endeavor was to substantiate the true diagnostic sign of mitral regurgitation; and also the significance of intra-ventricular or mitral non-regurgitant murmurs, as were held by my friend the late Dr. Cammann. He had learned by the most patient clinical observation, especially in his own case, in which the physical signs constantly reminded him of their progress to a fatal termination. He had also demonstrated, by pathological investigations, that the signs of mitral regurgitation as generally taught—murmurs at the apex beat, blowing, sawing, rasping, etc.,—were unreliable. That the true and invariable sign is a murmur of an entirely different character—a soft, rushing murmur, a blood-friction murmur, such as would naturally be formed by forcing fluids through an aperture, is heard behind, between the seventh and eight vertebræ of the left side, close to their spines. With this sign alone is mitral regurgitation certainly diagnosticated. The mechanism of the first sound is evidently the key to a correct diagnosis of a large majority of heart-murmurs, both functional and organic. The theories of the cause of the first sound, according to Bellingham, "may, for convenience sake, be considered, as the cause is supposed to be ex-

* Read March 18, 1875, before the N. Y. Academy of Medicine.

trinsic or intrinsic to the heart. Thus, under the first, it has been attributed to the impulse of the apex against the parietes of the chest; under the second head, it has been attributed to muscular contraction —in other words, to the successive shortening of the muscular fibres of the parietes of the ventricles. This is the oldest theory; it was adopted by Harvey, Haller, Senac, Bichat, and Corvisart. 2. To the sudden tension of the auriculo-ventricular valves. 3. To the friction of the blood against the parietes of the interior of the ventricles, or of the orifices of the large arteries. 4. To the collision of the opposite internal surfaces of the ventricles at the conclusion of the systole. 5. To the sudden elevation of the sigmoid and semilunar valves, caused by the wave of blood transmitted by the ventricles. 6. To the concussion of the blood transmitted by the systole of the left ventricle, with that contained in the aorta; and, lastly, to two or more of the foregoing causes combined."

I chose to consider as worthy of attention only three of the theories in vogue: 1. That of friction of the blood in its motion, within the ventricle, and its passage into the aorta. 2. That of the muscular contraction of the heart itself producing sound-vibrations, as shown by Dr. Wollaston, in 1810; and, 3. That of the vibrations of the mitral valve, caused by its closure and tension, and the forcing and rushing blood; and lastly, that some, recognizing the possibility of each of these three causes mentioned producing sound, have believed that, as the first sound is evidently composite, it is the result of all three. This was the one held by Dr. Cammann.

As none seemed to me to agree with all the conditions, and especially with the acoustical, I was impressed with the truth that none gave satisfactory evi-

dence of the cause of the first sound, and that we must direct attention to the heart itself for new proof on this vexed question. We find a peculiar musical-instrument arrangement within the heart, of a drum-like expansion of fibrinous tissue, to which are attached fine, tendinous cords, joining each part of the valve to the wall of the heart, through the intervention of bundles of muscular fibres—columnæ, carnæ, or musculi papillares. It seems incredible that such admirable conditions for producing sound-vibrations could have so long been overlooked by the many able observers, as the most probable cause of the first sound. That the first sound is caused by vibrations of the chordæ tendinæ, connected with the mitral valve in the left heart, and with the tricuspid in the right, set in motion by the swift current of forced blood, is a reasonable postulate, because agreeable to acoustic law. If this doctrine can be proved by pathological evidence of undoubted character, it simplifies our investigation.

If we can show cases of the mitral valve extending down into the ventricle like a funnel with a narrowed opening, the edges glued together and the chordæ tendinæ glued fast to the valve so that no vibrations could have taken place, and in which during life there were no murmurs nor a natural first sound, we may claim that we have demonstrated the cause of the first sound and of apex-beat murmurs.

The following cases are offered as supplying such convincing evidence:

*Case* I. *July* 6, 1859.—John Martin: Is a native of England; educated at Eton; forty-two years old; during the last ten years has been dissipated, and has had syphilis; had rheumatism eight years ago, which kept him in bed two weeks; and has since had frequent rheumatic pains; with these exceptions, has been well

until about two years ago, when his appetite failed, and he vomited mornings after taking beer; and his weight declined from one hundred and ninety to one hundred and forty-four pounds. Two days ago, while at his business, there was momentary loss of consciousness without falling, and similar attacks occurred frequently until last night, when they prevented sleep.

*Examination.*—The pulse grew gradually weaker, until it could no more be felt, and at the same time the respiration was suspended. The interval was so long, that I looked in his face to see if he were not dead; when, after a full inspiration, and a strong throb of the pulse, the heart would commence again and continue about fifteen pulse-beats, then cease, and begin again as before. In addition to this were attacks of "petite mal"—his face would flush slightly, and his eyes stare as if he saw a strange object—this would scarcely interrupt his conversation, when he would go on again as if nothing had happened. These epileptiform seizures came during the intermissions of the pulse and breathing, as well as at other times.

Auscultation of the chest discovered no fault in respiratory murmurs. There was slight systolic cardiac murmur, aortic-obstructive. After an intermission of the heart-beat, which agreed in length with the intermission of the pulse, it would begin again with a forcible impulse, which gradually decreased in strength until it ceased to be felt or heard, after which one contraction of the heart could be heard like a whisper, but without vocal sound and without impulse-beat. The sound of this contraction was peculiar; it was as if no blood was being forced into the aorta by ventricular contraction. By careful counting, repeated a number of times, the exact time of the heart's rest was

found to be sixteen seconds. The heart seemed to beat in a wild and peculiar manner, as if outside of the pericardium, and the point of impulse varied an inch or an inch and a half.

The next day Dr. T. M. Halstead was called as counsel, the conditions remained unchanged.

*8th.*—Was called at 6 A.M. to see the patient, who was supposed to be dying. I was informed that an intermission of extraordinary length had occurred. Respiration and pulsation had ceased, the hands fell by his side, his chin dropped, his head inclined to one side, and his face had become livid. His sister, who sat by him, believing him to be dying, called his wife; her outcries awakened him, and after a short time he recovered, and was as he had been before. When I arrived his pulse was twenty-five in the minute, as it had been from the first, and his state remained unchanged in both signs and symptoms.

*Friday*, 10*th*, 7 P. M.—Dr. Alonzo Clark was added to the consultation. Dr. Clark found the time of intermission of the pulse to be thirteen seconds; the seizures are a little more violent, and he is nervous. Physical signs the same as before.

11*th.*—Patient has slept during the night. The epileptiform seizures ceased at midnight, and the pulse has become regular without intermissions—fifty-two in a minute. After this the patient steadily improved, and one month afterwards he walked to Dr. Cammann's office in Fourth Avenue. Dr. Cammann diagnosticated systolic obstructive murmur, with hypertrophy of the heart, but believed the irregular action and peculiar symptoms were owing to functional derangement from indigestion. He became well enough to attend to business until October, 1861, when he was again taken ill. There were then anasarca, dyspnœa,

and laboring heart with obscure physical signs. He gradually failed, and died on November 26, 1861.

*Post-mortem* on 27th, assisted by Dr. Loomis. Complete adhesion of the pericardium to the heart. There was no free space, but in some parts the adhesions were stronger and apparently older than in others. The heart was largely hypertrophied, but was not weighed. The curtains of the aortic valve were thickened and shortened to incompetency, not holding water. The edges of the mitral valve were glued together, extending into the ventricle like a funnel: complete stenosis. The opening very small, the valve and chordæ were thickened and covered with plastic lymph, white and glistening.

*Case* II. (*Substance of Remarks made by* JAMES R. LEAMING, M.D., *before the Pathological Society on the Presentation of a Specimen for a Candidate for Admission.*)—Mrs. B——, twenty-three years of age, native of New York, widow, called Dr. S——, in April, 1869, for advice as to cardiac trouble and swelled feet. The doctor found, on examination, a systolic murmur over the base of the heart, more distinct over the aortic valves, gradually disappearing to the right in the course of the aorta; there was also a diastolic murmur.

*Diagnosis.*—Aortic obstruction and aortic regurgitation, with hypertrophy of left ventricle. There were also casts in the urine and albumen. She became dropsical, her condition gradually grew worse, and she died in September last.

I saw the case with Dr. S——, in May, and found no different conditions than those already discovered. *There was no mitral murmur of any kind.* The specimens here presented show Bright's small kidney of advanced disease. The heart is hypertrophied mostly in the left ventricle; the aortic valve is thickened at the

base of the curtains; shortened to incompetency — so far, agreeing with the diagnosis. But the mitral valve presents the most notable feature. There was no sign of disease of this valve during life, and yet it is damaged in a very peculiar manner. It is thickened by lymph-deposit; its color white, opaque; the edges of the curtain are adherent, and the orifice is narrowed down till it will barely admit the top of the index-finger; and the whole valve extends down into the cavity of the ventricle like a funnel. The chordæ tendinæ were shortened and thickened by lymph-deposits, and the musculi papillares were thickened and lengthened. But everything was symmetrical, viz., the funnel-like condition of the valve, the hypertrophy of the cardiac walls, of the musculi papillares, and of the columnæ carneæ.

With all of the conditions for producing a so-called *mitral direct murmur*, held by eminent authorities, there were neither mitral murmur nor first sound.

*Case* III. (*Copied from Reports of the Pathological Society, published in the Medical Record in* 1871.)—Dr. Loomis presented a heart, with the following history, from Dr. Milliken, house-physician of Bellevue: "Henry Clemens, admitted April 11, 1871, aged thirty-two; single; cabinet-maker by occupation; nativity, Switzerland. Patient gives hereditary history of pulmonary phthisis. Had an attack of articular rheumatism when seventeen years of age, from which he made a good recovery. States that neither at that time, nor since, has he experienced any precordial pain, but has noticed that after indulging in tobacco (for he has been an inveterate smoker) he would suffer from palpitation of the heart. He had had a cough, dating some time back, with some expectoration of a pearly white material, which he says he coughs up at night, at

which time his cough distresses him most. About two weeks ago, for the first time, he noticed that the sputa were streaked with blood. His cough remained about the same in character until one week ago, when he experienced a severe paroxysm of coughing, which was instantly followed by hæmoptysis, which continued for two or three days. Since the occurrence of hæmoptysis, he has had night-sweats, loss of appetite, depreciation of strength, and experienced a feeling of general *malaise*, and inaptitude for any kind of work; he complains also of insomnia and restlessness. His pulse is about 80, regular, but quite feeble; respiration somewhat hurried and easily performed. Heart: action regular, but *quite feeble;* apex beat on a level with nipple in fifth interspace. Heart-sounds *feeble;* after repeated examinations, no *murmurs could be detected.*"

The record proceeds to say that, while the patient was at dinner, he became suddenly unconscious and fell from his chair, and symptoms of paralysis continued until the 18th, when he died. *Post mortem* showed embolism of middle cerebral artery of left side, with softening of brain-tissue. Heart, fourteen ounces. Both right and left cavities contain large clot of blood; substance of heart relaxed: stenosis of mitral orifice only admits little finger; some shortening of chordæ tendinæ. The stenosis is due particularly to the thickening, shortening, and adhesion, of the chordæ tendinæ of the valve. The anterior portion of valve forms a bony mass, occluding that portion of the orifice. On the auricular aspect, the surface of the valve is ulcerated, the bony matter laid bare, and soft, reddish vegetations on the free border of the valve and upon the ulcerated surface. Pulmonary and tricuspid valves normal; little thickening at base of aorta."

Dr. Loomis remarked, "The case is of special interest, because with this marked stenosis no murmurs existed;" and Dr. Flint remarked that "the absence of murmurs might be accounted for—1. On account of rigidity of the valve not allowing a vibration; and, 2. The smoothness of the ventricular surface of the valve."

The first case, J—— M——, is full of instruction in its facts as regards functional disturbances of the heart and proof as to the mechanism of the first sound. The long period of rest, sixteen seconds, is worthy of attention. Observers who have watched the action of the heart in ectopia in an infant, as Cruveilhier, Bryan, and others, as well as when the heart has been exposed in experiments upon animals, tell us that the contractions of the auricles continue regularly, although the ventricles may be in a state of rest. And in this case no doubt it was so, notwithstanding that there was no first sound, no impulse-beat, and consequently no contraction of the ventricles. The importance of this fact cannot be over-estimated, because it invalidates much of the theory in vogue in regard to the causation of murmurs. It proves that the auricular systole may take place regularly, even when the auriculo-ventricular opening is very much contracted in stenosis of the mitral valve, without producing sound. Carefully listening under favorable circumstances after the last impulse-beat and first sound, one contraction, doubtless that of the ventricle, could be heard, without any vocal element of first sound, and was then followed by the long interval of silence, in which no contraction or sound of any kind could be heard.

The second case of Dr. S—— is a demonstration of the cause and mechanism of the first sound. "*There was no mitral murmur.*" With stenosis of the mitral valve, if the chordæ tendinæ had not been rendered in-

capable of sound-vibrations, by being plastered over with fibrinous deposit, there would have been a murmur, such as is usually heard in stenosis where the chordæ are free and uncovered. The first sound, and all murmurs connected with it, disappearing when the mitral valve and chordæ tendinæ are rendered incapable of sound-vibrations, is as convincing proof of their cause as is the experiment of hooking up a curtain of the aortic valve proof as to the cause of the second sound.

The first and second cases are confirmatory proof, that the cause and mechanism of the first sound, and the murmurs connected with it, depend upon the state and condition of the mitral valve and its chordæ tendinæ. In the second case there was no physical sign of disease of this valve during life, and yet it was found after death to be damaged in a very peculiar manner—thickened by lymph-deposits, opaque, its color white, the edges of the curtains adherent, the orifice narrowed down, barely admitting the tip of the index finger, and the whole valve extending down into the cavity of the ventricle fixed and like a funnel. The chordæ tendinæ were shortened and thickened, some of them glued to the valve, and the musculi papillares thickened and lengthened, as the wood cut from a photograph demonstrates. This case, during several months, was under the observation of the late Dr. Sprague, a careful and competent auscultator.

The third case, which is reported in the Transactions of the New York Pathological Society, is also confirmatory proof: "In the morbid specimen there was stenosis of mitral orifice—only admits little finger—some shortening of chordæ tendinæ. The stenosis is due particularly to the thickening, shortening, and adhesion of the chordæ tendinæ of the valve." During life,

heart-sounds feeble; after repeated examinations no murmurs could be detected. Could the proof be more conclusive?

The following experiments by Dr. Halford, quoted in the *British and Foreign Medico-Chirurgical Review*, April, 1860, is singular proof of the physiological cause of the first sound: "My proceedings were as follows: large dogs were obtained, and as in my preceding experiments (the animals being under the influence of chloroform), the heart was exposed and the circulation kept up by artificial respiration. A stethoscope being applied to the organ, the sounds were distinctly heard. The superior and inferior venæ cavæ were now compressed with bull-dog forceps, and the pulmonary veins by the finger and thumb; the heart continuing its action, a stethoscope was again applied, and neither first nor second sound was heard. After a short space of time the veins were allowed to pour their contents into both sides of the heart, and both sounds were instantly reproduced. The veins being again compressed all sound was extinguished, notwithstanding that the heart contracted vigorously. Blood was let in, and both sounds were restored. I have thus frequently interrogated the same heart for upward of an hour, and always with the like result."

The reviewer remarks: "There is an interesting circumstance which took place at one of Dr. Halford's experiments, which appears to us of great importance. It shows that when only a small quantity of blood finds its way into the ventricles, the first sound is still produced. The cavæ and pulmonary veins having been compressed, Mr. Lane, at whose request the experiment was performed, listened to the heart during its contraction, and said he heard the first sound indistinctly, not so clearly as before the compression. On

examination it was found that the vena azygos entered the right auricle by an independent opening, and was not secured: the vessel was compressed with the others, the heart contracted, no sound was heard."

This experiment proves that the contractions of muscles of the heart give out no sound; for without blood moving through the heart there would be silence. When there was no blood forced there was no sound; and we have just shown, by pathological specimen, that when the chordæ tendinæ were rendered incapable of vibration, there was also neither sound nor murmurs. Consequently, the first sound and murmurs must be the result of chord and valve vibrations set in motion by the rushing blood. The blood is the bow applied to the strings to give vibrating sounds; and murmurs are sounds of individual chord-vibrations not in unison.

One of the points I endeavored to establish in 1868 was that the presystolic murmur, called also the auricular-systolic and the mitral direct, is one of the intra-ventricular murmurs, caused by vibrations of chordæ tendinæ subjected to irregular tension, and not by blood being forced through the contracted opening of the mitral valve in stenosis. Although frequently connected with that pathological condition, it is yet oftener an accompaniment of pathological change of the mitral valve without stenosis. My argument was, as Dr. Cammann first contended, that the auricle was too feeble a power to force blood through the contracted opening of the diseased valve, so as to cause sound which may be heard through the chest-wall, even if empty, much less so when the ventricle is filled with blood; and, lastly, I maintained that the murmur does not agree in length with the time of contraction of the auricle. According to the best authorities, the con-

traction of the auricle is instantaneous, while the murmur is of considerable length.* If contraction of the auricle could cause the murmur, the two ought to agree in time. According to Bellingham, "the systole of the auricle is a quick, short, sudden motion." Lower says, "Its rapidity equals the explosion of gun-powder, and immediately precedes the ventricular systole, the one motion appearing to be propagated by the other."

Marey assigned to it two-tenths of the time of the heart-beat, which is many times longer than the reality, and yet is much shorter than the time of the so-called presystolic murmur. This murmur, too, has none of the qualities of sound which should be produced by blood forced through a narrowed opening in the valve. But all argument becomes unnecessary in presence of the foregoing pathological facts and clinical history. Dr. Frank Donaldson, Professor of Physiology and Hygiene, and Clinical Professor of Diseases of the Chest and Throat, University of Maryland, in a paper read before the Medical and Chirurgical Faculty of Maryland, annual session, April, 1874, on "Significance of the Presystolic Murmur," relates the following cases, with remarks:

"Some years ago (in 1867) a case came under my observation, which made me question the explanation which I had adopted on the authority of Barth, Roger, Walshe, and Flint, of the sound which was described first by Fauvel, in 1843, and then by Grisolle, as the presystolic murmur, afterward by Dr. Gairdner, of Edinburgh, as the auricular-systolic murmur, and by Dr. Austin Flint as the mitral direct murmur.

"These authorities claimed that this sound was heard just *preceding* the ventricular contraction, and was

* Harvey, Lower, Bellingham.

caused by the systole of the auricle forcing the blood into the ventricle, through a diseased and contracted auriculo-ventricular orifice.

"The case was a man sixty-four years of age, of grossly intemperate habits, who came to the Baltimore Infirmary with symptoms of advanced heart-disease, great dyspnœa, a small, contracted pulse, heart much hypertrophied, with a murmur of a rasping character, heard loudest between the second and third ribs at the base, not extending up the carotids, but down toward the base, and completely obliterating the second sound of the heart. The murmur was audible after the apex beat and the systole of the ventricle, and was followed by the pause of the heart. The first sound of the heart was normal. The diagnosis seemed clear and unmistakable, and was recorded as insufficiency of the aortic orifice, by means of which the arterial blood was forced back into the left ventricle.

"The *post mortem* showed atheromatous degeneration in the aorta above the semilunar valves extending to the sacs of Valsalva, and causing adhesion of one of the semilunar pouches of the aortic orifice to the wall, so bending it down that that portion of the orifice was unprotected. The second sound could not be produced, and the insufficiency of the valve was evident.

"Thus far the diagnosis was correct, but on examining the mitral orifice we found, to our surprise, that it was reduced by thickening at its base to about the size of one quarter of an inch in diameter. Yet, during life, there was no abnormal sound preceding or during the ventricular systole. With such a contraction of the left auriculo-ventricular orifice, ought we not to have had a decided presystolic murmur? The whole heart, auricle and ventricle, was enlarged and increased in force, and yet there was no murmur produced from

the passage of the blood through an orifice so reduced in size! I could not help questioning the received opinion as to the significance of the so-called mitral murmur. As it is a physical sound, heard at a particular period of the heart's action, the physical cause which was said to produce it being present, it ought to have been heard, but it was not.

"Hope, as far back as 1842, reports a case where the mitral orifice was so contracted that it would only admit the little finger, yet there was no murmur during life, preceding the first sound. In his report he adds: 'I have frequently known a contraction of the mitral orifice to the size of only two or three lines, to occasion little or no murmur.' Dr. Stokes, in his work on 'Diseases of Heart and Aorta,' relates two cases of extreme contraction of the mitral orifice found after death, but where, during life, there had been no murmur audible even to his practised ear.

"Dr. Waters. His first case was where he heard a loud systolic as well as a presystolic murmur. At the autopsy there were found insufficiency and slight contraction of the mitral orifice. In the second case there was no presystolic murmur whatever, although the autopsy showed a constricted mitral orifice only admitting the tip of the index-finger. Next follow the details of four cases of extreme contraction of the mitral orifice, where, during life, there was no presystolic murmur audible. He candidly adds: 'I have given you instances sufficient to prove that great constriction of the mitral orifice may exist without there being any murmur produced by the passage of the blood from the auricle into the ventricle, and therefore that you must not look for a mitral-diastolic or presystolic as a constant sign of obstructive mitral disease. My belief is that this murmur is far more frequently

absent than present, even when there is great obstruction at the mitral orifice.' Dr. Waters accounts for the presence or absence of this murmur, as depending on the greater or less vigor with which the auricle contracts."

Dr. Donaldson sums up his relation of cases and remarks: "Thus we have eleven cases of the lesion without the murmur, and three cases of murmur without the lesion" (quoting the latter from Dr. Flint).

The diagnostic sign of mitral regurgitation, which has been and is still taught, is a harsh, blowing, sawing, or filing murmur, heard during the systole at the apex beat. Upon the accepted authority of this murmur, which is so often met with, the great frequency of mitral insufficiency has come to be considered as incontrovertibly established.

The cases we have already related are proof that these murmurs are not heard when the chordæ tendinæ and valve are rendered unfit for sound-vibrations. J. S. Bristow, M.D., London, F. R. C. P., Physician to St. Thomas's Hospital, in an article on "Mitral Regurgitation, arising independently of Organic Disease of the Mitral Valve," in the July number of the *British and Foreign Medico-Chirurgical Review* of 1861, gives six cases, with introductory remarks. His arguments and points in the cases, show that instead of proving that regurgitation may take place through the mitral valve without disease, they in reality disprove the theory in vogue, and confirm the doctrine of chordæ-tendinæ vibrations as cause of the first sound.

Dr. Bristow remarks: "It may almost be regarded as an axiom in medicine that the presence of a systolic apex murmur is positive proof of regurgitation through the mitral orifice. I have not hesitated to adopt it in

reference to the cases already detailed." The following are quotations from his cases:

*Case* I.—There was a distinct systolic murmur audible at the apex of the heart.

*Post mortem.*—The aortic and mitral valves were perfectly natural.

*Case* II.—There was an increased area of dullness in the cardiac region, and a systolic bruit loudest at the apex of the heart.

*Post mortem.*—The muscular tissue and the valves appeared perfectly healthy.

*Case* III.—The impulse was diffused and heaving, but not very strong. A systolic murmur was detected at the apex of the heart.

*Post mortem.*—All the valves were healthy-looking.

*Case* IV.—First sound at the apex was flapping and prolonged.

*Post mortem.*—The valves were perfectly healthy in texture.

*Case* V.—The cardiac dullness was enlarged, and a systolic murmur was audible with the heart's action, most distinct at a point an inch below, and internal to the left nipple.

*Post mortem.*—All the valves appeared perfectly healthy.

*Case* VI.—There was a distinct but not very loud systolic murmur, loudest in the usual situation of the apex of the heart.

*Post mortem.*—The aortic and mitral valves were perfectly healthy-looking, and doubtless quite competent.

The proof is conclusive, if a multitude of cases prove anything, that mitral *stenosis* is not necessarily accompanied by an apex-beat murmur.

A tabular arrangement like the following, in classifying murmurs acoustically, may be useful:

| | |
|---|---|
| Valvular (all organic.) | Aortic obstructive systolic. |
| | Aortic regurgitant diastolic. |
| | Mitral regurgitant systolic. |
| Intra-ventricular (more or less functional]. | Organic functional. |
| | Inorganic functional. |

These two great divisions are made in accordance with their acoustic differences. The sound in valvular murmurs, is a friction-murmur, that of blood forced through an aperture. The intra-ventricular murmurs are mostly and distinctly chord-vibrations. The contraction of the muscular walls of the heart and its fleshy columns, the friction of rushing blood among the chordæ tendinæ and against the tense mitral valve being the occasion of sound vibrations, but is not the mechanism of the sound itself. As great difference exists between these murmurs as between that of a whisper and that of the voice. The obstructive systolic aortic may be modified by irregular calcifications in the aortic valves, extending into the column of forced rushing blood. In this way a harsher character may be given to the murmur, or it may even become musical. Vegetations also attached to the orifice or valve may be thrown into vibrations in the column of blood, and produce a musical murmur, but these are rare, mere possibilities. When musical murmurs occur they are almost always, vibrations of the chordæ tendinæ, some of which are under extraordinary tension.

These sounds and murmurs may be illustrated by a stringed musical instrument. Every degree in quality of murmur or sound, from the softest blowing up to the harshest, sawing, rasping, filing, or, when the vibrations become sufficiently rapid and regular, into musical sounds. The use of the term "bellows sound" by

Laennec was unfortunate as applied to the murmurs of the heart, and much of the misunderstanding of murmurs and their mechanism is due to it. It is true that it describes the friction-murmur of blood forced through an aperture as in aortic regurgitation. It is like the sound of the air forced through the bellows; but the bellows-sound is not so like the friction-murmur of blood forced through an aperture as is fluid forced through an elastic syringe, in which some obstruction is created by pressure upon the tube. But, to imitate the murmur exactly, a fissure should be made in the bulb of the syringe, and then compressing it with force, the fluid escaping will give the exact sound. The only friction-sounds in cardiac murmurs proper are where the blood is forced through apertures or past obstructions; it is heard at the aortic orifice when there is obstruction, as by lymph-deposits upon the valve. It is at first uncomplicated, the simple gushing sound. But in time the obstruction causes hypertrophy of the left ventricle, which having taken place, irregular tension of the chordæ tendinæ is the result, and vibrations out of unison with the first sound are carried with the current of blood, and both occurring in the systole are mixed together and form what is called the blowing murmur.

It becomes a sound of mixed elements, friction of blood against a solid, and vibration of strings under irregular tension. In order to have an intelligent understanding of these murmurs we must analyze them and separate the sources of sound. We are assisted in this by localizing the sources. The blowing, sawing, filing, rasping sounds have their origin and cause within the ventricle; they are intra-ventricular. Dr. Cammann called them mitral-non-regurgitant. They are

heard over the base of the heart, but always with greatest intensity at the apex beat.

Friction-sounds are heard best over the orifices or in the direction of the vibrating column of blood. The aortic systolic obstructive murmur is heard over the aortic valves, and in the course of the column of blood. The regurgitant aortic diastolic murmur is heard over the aortic orifice, and to the left and toward the apex beat. The mitral aortic-regurgitant is heard behind on the left side near the spine. In this direction the blood is forced in regurgitation through the mitral valve; impinging first against the auricular wall, lying against the œsophagus, and aorta, and inter-vertebral substance, it is conducted directly to the ear, giving the sensation of being shot into it.

It may be heard a short distance from this point conveyed through the chest-wall. It may be heard in front, at the apex beat, by conduction through the substance of the heart, when there are no intra-ventricular murmurs to destroy it or take its place. The discovery of this absolute sign of mitral regurgitation belongs to Dr. Cammann, and his last professional thought was given to its consideration. It is one of the most certain of cardiac signs. This characteristic murmur, heard in the situation he has pointed out, is an unfailing sign of mitral regurgitation. It had been my opinion that this characteristic murmur was never heard in front at the apex beat—as it certainly is not when the valve is diseased, and the loud intra-ventricular murmur drowns and supplants it.

But the following case shows that it may be heard both behind and before in congenital mitral insufficiency, without hypertrophy of the heart and without lymph-deposits upon the valve.

*Case* VII. (December 12, 1870.)—W. S. R., New

York, aged twenty-two; mason, living in Yorkville; is a fireman temporarily, and was a member of the old fire department. Has never been sick, except with chills and fever. Sent for examination by Dr. Charles McMillan, surgeon of the department. There is a systolic murmur at the apex beat accompanying the first sound; it is a soft, gushing murmur, and can be heard in the chest-wall more to the left than to the right side. It is heard also with directness and greater intensity between the seventh and eighth vertebræ, left side behind, near the spine. The murmur is shot into the ear when it is placed over this point. It can be heard some distance to the left, conveyed through the chest-wall. It can also be heard over some portions of the right lung posteriorly, at the inner angle of the scapula; also at the lower angle, being a faintly conveyed sound.

One year after, examined him again. Signs unchanged. This murmur has the same quality in front as behind. It has none of the vocal element of apex-beat murmurs, usually described as diagnostic of mitral regurgitant murmurs. Yet I have no doubt that this murmur is caused by mitral insufficiency, which is congenital, without hypertrophy of the heart, and without disease of the mitral valve.

A great majority of cardiac murmurs, even of those accompanying organic disease of the heart, are in a manner functional. That is, the murmurs are not organic in the same sense that the valvular murmurs are; which are organic murmurs because the structural change in the valve is part of the mechanism of the murmur. Intra-ventricular murmurs, even when the result of structural change in the heart, may be considered functional, inasmuch as that they have their mechanism in vibrations of the chordæ tendinæ, which are themselves unchanged by any diseased action, but

simply vibrate, giving out sound of high or low pitch, soft or harsh, feeble or loud, according to the degree of tension of the individual strings, and the force of the heart's contraction. The cause of irregular contraction of the heart-muscles may be from disturbed nerve power, as well as from organic change.

Functional murmurs proper may occur in the healthy heart, are transient, passing away with the subsidence of the cause, which may be anæmia, hyperæmia, sympathy with brain-disease, stomach, liver, or it may be from disorder of the nervous system, the influence of tobacco, coffee, tea, or any narcotic or stimulant having influence upon the organic life of the body, of which the heart is the centre and citadel.

Functional murmurs do not signify danger of sudden death, but nothing more alarms patients than disturbed action of the heart. When the heart seems to stop, and then to turn over and thump against the chest-wall, the sensation is not a pleasant one, even to a medical philosopher. It is no wonder that it creates intense alarm in the lay patient, especially if accompanied by prolonged palpitation or faintings.

These conditions may be the forerunner of softening, or fatty degeneration, but they signify always that there is over-distension of the portal system. Intermissions of the heart-beat and pulse may be present for years, and be merely the result of functional disturbance from chronic indigestion.

Intermissions of the pulse are popularly considered as signs of heart-disease. Life-insurance companies, in printed forms, make it the duty of examiners to reject as unsafe those who have intermittent pulse. It is possible that this rule militates against the interest of the companies, and it certainly is a source of great alarm and possible damage to the rejected applicant.

The sign, of itself, is no proof of heart-disease, but is proof of indigestion, or pericardiatic adhesion to the lungs or heart. It is true, cardiac disease is frequently a cause of indigestion, and thus, secondarily, the cause of irregular pulse. But a confirmed dyspeptic is usually a safe life, for he is not likely to commit indiscretions in diet, as he is continually warned to desist by functional disturbances. Proper medication will generally relieve intermittent pulse, even in advanced cases of cardiac disease.

A sedative dose of calomel will frequently set it right at once, and the intermissions will disappear.

The late Dr. Samuel Henry Dickson stated that, during the first hours of sleep, children have intermittent pulse, which will disappear when they are awakened. This is true, especially with those children who are allowed over-stimulating food, but, as the night passes on, and the food becomes digested, the intermissions cease. In the adult, the occasion of a wine-dinner, with tobacco, is often followed by intermittent pulse, especially during sleep, when the circulation is sluggish.

The cause of the rhythmic movements of the heart is debatable ground. That it is within the heart itself can scarcely be questioned, for, when the heart of some animals is dissevered from all connections, and taken from the body, it may go on performing its rhythmical movements. Still, the quality and quantity of blood influence them in an unmistakable manner. The fact that shutting off supply of blood to the structure of the heart will arrest its contractions was shown in 1842 by Mr. Erichsen. Dr. Brown-Séquard has attempted to explain the motion to be due to the carbonic acid present in the venous blood, and Dr. Radcliffe has also given a similar explanation.

The experiments of Dr. Paget show that the power

causing rhythmical motion does not reside in all parts of the heart alike; that, in fact—

"If, for example, the cut-out heart (of any of the amphibia) be divided into two pieces, one comprising the auricles and the base of the ventricle, the other comprising the rest of the ventricle, the former will continue to act rhythmically, the latter will cease to do so, and no rhythmic action can be by any means excited in it. The piece of ventricle does not lose its power of motion, for if it be in any way stimulated, it contracts vigorously, but it never contracts without such an external stimulus, and when stimulated it never contracts more than once for each stimulus.

"Other sections of the heart, and experiments of other kinds, would show that the cause of the rhythmic action of the ventricle, and probably also of the auricles, so long as they are associated with it, and not with the venous trunks, is something in and near the boundary ring between the auricles and ventricles; for what remains connected with this ring, or grew with a part of it, in a longitudinally bisected heart, retains its rhythm, and what is disconnected from it loses its rhythm."

If we take a merely material view of the subject, no doubt we have arrived at the solution as nearly as we ever will. But is it useless or absurd to look further? The experiments of the great Harvey with the egg of the hen show that active life remains inchoate in the *punctum saliens* or germinal spot until warmed into active life. This principle came into the egg organization at the time of its fecundation. Its first life-motion is rhythmical movement of particles before any portion of the heart's structure can be seen. The little red point appears and disappears rhythmically, and thus the *principle* builds its house, the auricle being its first

chamber. The very nature of this principle is rhythmical. Its special home is in the ganglionic nervous system, but it pervades the whole body; wherever there is nerve-fibre accompanying the smallest capillary —the vaso-motor—it is present. Aberration from its normal life-action is disease; and influences, both outside and inside of the body, make impressions upon this life, helping to determine the character of the disease. Medicines act upon it, but their *modus operandi* is a sealed mystery. That they are purgative, emetic, stimulant, sedative, or alterative, we only know the fact. The heart, supplied with about three hundred ganglia, is the centre and citadel of this life, and its abnormal or disturbed action is sometimes mysterious evidence of both intrinsic and extrinsic disease.

Acoustic properties of the chest have not been dwelt upon as their importance demands. The diagnosis of murmurs within the chest is facilitated, or otherwise, according to its conditions as an acoustic chamber. The difficulty of hearing signs in the chest of a hunchback is recognized; it is also a well-known fact that, as the heart enlarges, the murmurs grow weaker, so that those which had been once easily detected become feeble, or disappear altogether. Still they have been accounted for, it seems to me, upon every other principle than the true one.

In Dr. Cammann's last illness, by his request, I was called to examine him. After he had explained to me that I would find obstructive and regurgitant murmurs, of which he had been long cognizant, and of which he explained the cause and origin, and of their gradual increase, I found that I could but just hear the soft, feeble murmurs of aortic obstruction and regurgitation, but intra-ventricular murmurs were not heard. I told the doctor that the regurgitant murmur which he had

emphasized in relating the case was slight: "Yes," he said, "it is but a chink." Dr. Peugnet told me that when he examined him at the beginning of his illness the murmurs were loud and easily heard. I felt mortified that my ear had failed me, as I supposed, caused by a long ride in the cold, in an open carriage. The doctor had circumscribed pleuritis with effusion and pneumonia. In time the effusion was absorbed, and then the murmurs at the apex beat were easily heard.

Another case, of which I have no notes, in which I failed to make out a murmur where it should have been heard, and which afterward returned, as the intercurrent pneumonia, became convalescent, also annoyed me, and again I blamed my ear. Not long afterward I saw in the London *Medical Times and Gazette* or in the London *Lancet*, the question "Why do cardiac murmurs disappear during pneumonia or pleurisy?" I felt at once that the cause of my not hearing the murmurs more plainly in Dr. Cammann's case, as well as in that of this other patient, was because they were obscured by some cause I then did not know.

Other cases of cardiac murmurs disappearing or becoming obscured during the presence of pneumonia or pleuritis led me to believe that it was in accordance with physical law. A patient with pleuritic effusion was sent to me by Dr. Otis for examination. I knew from a previous auscultation that he had aortic obstructive and aortic regurgitant murmurs. At this time, however, they could not be heard. I wrote to Dr. Otis, stating these facts, and predicting that when the effusion was absorbed these murmurs would again return, which proved to be the case.

On August 27, 1864, I saw Miss Hall, matron of the Home for Soldiers' Children, in Fifty-seventh Street near Eighth Avenue, with Drs. Charles McMillan, J. L.

Smith and E. Krakowizer. There were no heart-murmurs, but as all the rational signs of cardiac disease, with increased area of dullness under percussion, signified hypertrophy, it was suggested that we should examine her for pneumonia, and, upon raising her up and listening behind, it was clearly made out. I then predicted that, when the pathological results of pneumonia were removed we would be able to diagnosticate her cardiac disease. This was afterward done, and Dr. J. L. Smith took notes of the examination, and upon her death, some months afterwards, was able to verify the diagnosis. He presented the heart, with history, to the Pathological Society, and a committee was appointed to examine into the facts concerning the disappearance of heart-murmurs during the presence of pneumonia and pleuritis, and to report. If my memory serves me, the committee reported in substance, in the summer of 1865, that in some cases observed in Bellevue Hospital, murmurs grew feeble or disappeared on the advent of pneumonia or pleurisy, but that it was the opinion of the committee that this phenomenon was owing to the feebleness of the heart and its frequency, for in the cases noticed the pulse was 120 or more per minute.

These reasons I had myself considered and rejected, for at the same time that Miss Hall was ill I had another patient, O. B. H——, who had had for years a double aortic murmur, which, when he was attacked with pneumonia, disappeared. His pulse ordinarily was about fifty in a minute, but during the pneumonia it rose as high as eighty, but no higher. Drs. Charles Mc Millan and J. L. Smith were also both cognizant of the facts as narrated. The philosophical explanation of these phenomena occurred to me during the winter of 1864–'65, with

the following proof and illustration. The chest is a musical chamber, and may be represented by a violin. When the instrument is tuned and in order, its acoustic qualities may be considered as perfect. If a watch or music-box be placed within the violin, auscultation will reveal the slightest jar or noise made by the works of the watch, or bring out with distinctness the low tones of the music-box. But if, while the ear or stethoscope is still placed upon the violin, water or sand be poured into its chamber, the sounds of the box or watch will grow feeble or disappear. The low notes of the music-box disappear entirely, as also does any jarring of the wheels of the watch. These phenomena are invariable because they are the result of acoustic law. Scientific medicine is the immediate professional want of our time. If acoustic law be applied to auscultation in physical diagnosis, it will remove it from the domain of doubt or uncertainty.

# PART III.

## ARTICLE III.

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### DIAGNOSTIC AREAS UPON THE CHEST WALL

HEART sounds are natural and indicate a normal condition of the heart. Heart murmurs are not natural and indicate an abnormal condition of the heart, and are diagnostic of structural or functional disease. These are elementary truths and yet must be borne in mind, for in practical application there is an endless variety which may leave us in doubt or in error unless we refer back to them.

We can imagine a true picture of the heart and lungs acting without any intervening chest wall. Placing the distal end of the stethoscope immediately against the heart over the source of sound, for instance over the ventricle or over the aortic orifice, we note the normal first-sound accompanying the systolic contraction of the heart. We notice that the first sound is formed within the ventricle and is heard over every part of it from which ever side we make our examination. That the sound goes *with* the current of blood *into* the aorta and passes along into the arteries until it is lost in their minuter divisions. Now if we examine the same heart in its normal position within the chamber wall, we will notice that we only have direct conduction of sound at the base and apex beat, and we rightly say that we hear the first sound over

the base of the heart intensified at the apex beat. At these points the heart comes against the chest wall, which conducts the sound into the circumscribed areas of contact; from thence it is spread in diminished intensity over a larger space until it is lost entirely. Functional murmurs are heard in the same areas under the same limitations.

But frequently adhesions of the pericardial sac to the lung and the lung to the chest wall will form direct conduction into limited areas above or below, or to the right or to the left of the apex beat and some distance removed.

This condition may give us the peculiar phenomena of a distinct murmur over the area of adhesion, with normal apex beat. The only pathological condition is the sound-conducting adhesion, which phonates the natural interior sounds of the heart which are not heard otherwise, and which being brought into the chest wall produces a simulated blowing murmur. This gives rise to errors in diagnosis, the murmur being mistaken for the sign of mitral regurgitation which the books teach us to look for in this area, but which in reality is behind and never in front as a direct murmur, which this is. The error may be corrected in this way. The apex beat is not changed in location. There is no direct murmur at the apex beat, and the rhythm is unaffected. Another correction is that the treatment may cause the removal of the plastic exudation by which the sound is conducted and the murmurs will then cease. But this takes some time. In all of these cases above supposed, the apex beat and the rhythm are not changed, which are solid reasons for believing that the causes were extra cardiacs.

We pass to the consideration of another class of murmurs also heard at the apex beat, because there

the sound is brought into the chest wall by impact of the heart from within. These murmurs are of a harsher character than the blowing, and they imply structural change within the heart, and are followed in a short time by change of the area of apex beat, being removed to the left with a perceptible loss of rhythm, and of the harmonious quality of the first sound. Then we know that the heart is hypertrophied, and this denotes structural change within the heart, which threatens the life of the individual.

I have used this illustration to show the method by which I propose to continue the study of heart sounds and murmurs. In practice we are obliged to study the heart sounds and murmurs through the chest wall chamber, subject to limitations and alterations which may mislead us as to the truth. But if in the mental picture which we are obliged to make in *all* cases, we may correct and complete it by examining the heart's action and sounds and murmurs without any intervening chest walls, and apply the laws of acoustics and mechanics, we come directly to the truth.

We can seldom do this by direct experiment, but we can do it practically by applying the unvarying laws of exact science.

Examining the heart's sounds and murmurs over and through the chest wall, we must remember that the points of contact or attachments of the heart sac to the chest wall constitute diagnostic areas.

They are windows through which the mind looks into this sound chamber, as it were, and sees the secret action of the heart.

### Area of the First Sound.

The first sound is heard over the ventricle with greatest intensity at the apex beat. This is the area,

too, of most of the functional murmurs, and of all of the intra-ventricular and mitral non-regurgitant. But as the heart is not permanently fixed in its position, a variation of areas of intensity may occur without any structural change of the heart itself, as from being crowded from its place by effusions or morbid growths. Inter-pleural adhesions frequently draw the heart from its normal position without altering its normal function, dislocating the heart and conducting sound-vibrations into new areas, complicating opinions and constituting an endless source of mistaken diagnosis.

When adhesions happen to take place in such a way as to contract in a certain direction—upwards for instance—the heart and lungs together may be drawn upwards until the apex beat may be felt and heard with greatest intensity between the fourth and fifth ribs.

Or the adhesions may draw the heart in other directions, changing the area of sounds and murmurs.

Again the internal organs may not be displaced by any interior force, but contraction of the chest-wall may bring it in contact with the heart and thus create new areas of sounds and murmurs. The areas of cardiac murmurs have greater variation than the areas of sound. Genuine valvular murmurs denote structural change, which is progressive and is followed by hypertrophy.

As the heart enlarges the areas change. Or adhesions attaching the pericardial sac to the lungs and the lungs to the chest wall phonate the murmurs into new areas. Or, as sometimes happens, phonate the normal heart sounds into definite areas, simulating cardiac murmurs.

So it is readily seen that all the rules laid down in text-books for diagnosis of murmurs at the apex beat or near it, instead of being sure must have a very

elastic interpretation, if they do not cease to be rules at all.

But the areas of obstructive aortic and of the mitral regurgitant murmurs of Cammann, are more definite and fixed, and consequently are of more importance in diagnosis.

The area of aortic obstructive systolic and the area of aortic regurgitant diastolic murmurs have common points of commencement at the aortic area or the area of the second sound, that is, over the aortic valve.

The obstructive extends across the sternum, or under it rather, and is intensified to the right of the sternum over where the aorta emerges from under it, and just before it dips backwards under the lung in forming the arch.

The area of regurgitant aortic diastolic commences at the same point over the aortic valve, and extends in an opposite direction, that of the regurgitant stream of blood. If we could remove the chest wall the murmur would have one invariable direction when examined with the stethoscope, viz., that of the stream of blood into the ventricle which at that time would be empty and relaxed. But by conduction into the chest wall it is frequently varied according to the contact, and may be heard not only over the ventricle but even down to the apex beat. Sometimes it is heard only in the middle of the sternum, an inch and a half below the aortic valves.

These variations are due simply to conduction of sound, that portion of the heart in which the murmur generates coming in contact with the chest wall or the sternum at different points.

The area of mitral obstruction murmurs, non-regurgitant murmurs, intra-ventricular murmurs, and the so-called mitral regurgitant murmurs of Bellingham

and succeeding writers, also the mitral direct of Flint and others, extends from the apex beat to the left, sometimes to the angle of the ribs behind, sometimes to the right as far as the sternum. This area includes murmurs of great variety as to their cause and significance, but in all, the greatest intensity of the murmur is at the apex beat, except where the heart's sounds or murmurs are phonated into definite areas outside of the area of apex beat. The area of the first sound and the area of the intra-ventricular murmurs are the same, except in advanced organic disease of the heart. The mechanism of each is in the contraction of the heart over a ventricular cavity filled with blood, which it forces out into the aorta. If the heart is normal and the contraction is normal, perfect first sound is the result. But if the heart is irregularly hypertrophied or dilated, or if there is disease of the mitral valve or deposits of lymph upon it or upon the chordæ tendinæ, then instead of the harmonious first sound, we will hear discords.

If there are deposits upon the mitral valve, or if it is thickened, or if its free edges are adherent in part, or attached to the chordæ, or if some of the chordæ are thickened or shortened, or made tense irregularly by spasmodic contraction of some of the muscles of the heart, there will be unusual sound vibrations denoting the cause of the discord, which may be accurately interpreted by the experienced auscultator.

Occasionally the apex beat itself, with all its varying murmurs, the so-called mitral direct, and mitral regurgitant of writers, with all the rest, disappear from the front entirely. What has become of them?

This simple explanation will tell: Adhesions attached to the posterior wall of the chest contracting, withdraw the heart from the front chest wall, and, break-

ing the sound connection, of course all murmurs cease. This region has thus become the fable land of theories and fanciful diagnostics.

Also the pulmonic area over the pulmonary artery and the left auricle is sometimes the region of murmurs, which have given rise to different interpretations, mitral regurgitation, or pulmonic regurgitation, and quite a little controversy. If we direct our attention to the anatomy, we will find that normally there is quite a depth of lung tissue over the heart and arteries at this point. Normal lung tissue is a non-conductor of sound. And in health there are no murmurs over this area, but effused lymph, or plastic exudation, may result in making sound connection with the chest wall; and whether there are regurgitations through the mitral valve or through the pulmonic valve or not, murmurs will be heard there wherever these adhesions are attached to the chest wall.

They have no other significance, and are to be classed with other accidental areas made by adhesions over the front, or of any other part of the chest.

### Cammann's Area.

The true mitral regurgitant murmur is only heard behind, to the left of the spine, and near it, with maximum intensity between the 7th and 8th vertebræ. This, like the areas of aortic obstruction and aortic regurgitation, is fixed and invariable. It was first pointed out by the late Geo. P. Cammann, M.D., of this city. It is a characteristic murmur like the aortic regurgitant, and like it is formed by blood being forced through an aperture in a valve imperfectly closing the auriculo-ventricular orifice. The stream of regurgitant blood strikes upon the side of the auricular wall

which lies against the aorta and the œsophagus, and the sound is transmitted by direct conduction through the inter-vertebral cartilage between the 7th and 8th vertebræ near the spine. It is absolutely diagnostic, and when it is heard there is always incapacity of the mitral valve. There are no exceptions. The murmur is sometimes heard by indirect conduction as high up as the upper border of the 7th, and as far down as the lower border of the 8th, but the greater intensity is always heard over the intervening inter-vertebral cartilage. If we could remove the vertebræ and chest wall and place the end of the stethescope upon the side of the auricle, here alone is where we would hear the murmur in its directness and greatest intensity.

I have named this diagnostic space, Cammann's area, which, like the aortic obstructive and the aortic regurgitive areas, is more fixed and more diagnostic than the intra-ventricular area in front, and is of far greater value in diagnosis. If the chest wall were removed we might place the stethoscope against the right auricle, and hear the tricuspid regurgitation where it exists, or over the pulmonic orifice and hear the pulmonic obstruction and regurgitation; but practically they are covered by healthy lung and their murmurs are not transmitted to the chest wall, and they are never heard except through pathological results, such as tumors, or consolidation of lung tissue, or by extensive adhesions which may conduct the murmurs into the chest wall, but without definite areas and rules for diagnosis.

In summarizing we may say that there are four natural diagnostic areas on the chest wall. The aortic obstructive, the aortic regurgitant, have the aortic orifice as a point of union and departure, and Cammann's area of mitral regurgitant murmur behind,

between the seventh and eighth vertebræ, are fixed and practically invariable. The mitral non-regurgitant or intra-ventricular area, may be more or less changed by dislocation of the heart, which is frequently the case.

The heart is a dual organ, the left and the right. There are two circulations, the systemic and the pulmonic. Each heart has two cavities, the auricular and the ventricular. Each cavity has a main door of entrance and of exit. Each door or orifice of the ventricle may have two murmurs, the obstructive and the regurgitant. Obstruction at the mitral orifice is shown by the mitral non-regurgitant murmur.

The murmurs of the right heart are, no doubt, the same as the left, but they are lost in the non-conducting lung tissue, and are never brought to the chest wall except by accident.

# PART III.—MISCELLANEOUS.

## ARTICLE I.

### Therapeutics of Chloride of Ammonium.

Sal ammoniac, muriate of ammonia, hydrochlorate of ammonia, or, properly, chloride of ammonium, are designations of the salt some of the remedial powers of which I propose to consider in this paper.

Our pharmacopœia presents us with a variety of medicinal agents, and each has its measure of power; each acts in a specific way peculiar to itself or its class upon the living organism, and is beneficial, or otherwise, according to the wisdom of the practitioner directing its use.

Our knowledge of therapeutics is mostly empirical; *a priori* reasoning has little to do in determining our choice of agents; a knowledge of their intrinsic value is *approached* only after many trials by different observers under many and different circumstances. In this view, it may be asserted that all the remedies in common use are still upon trial. In endeavoring to estimate the value of a remedy by the light of experience, in order to prevent hasty conclusions, it is well enough to premise that many of the sick calls any practitioner may attend are either wholly imaginary, or of that class of diseases called functional, in which the "*medicatrix naturæ*" is frequently competent to perform a cure, especially when stimulated by the imagination; but that when a material, potent substance is requisite to remove a morbific cause, or impress a vital change upon the system, the domain of fancy ends, and that of material facts takes its place.

The idea that all medicines are still and ever must be on trial till we have arrived at perfection in our knowledge of therapeutics is illustrated in opium. How long has the poppy been the sweet soother of pain and care, giving balmy sleep to the wearied, excited brain, and rest to the tired limbs; when fever rages, and every fibre of the body is quick with anguish, how blessed is the repose it gives, how delightful the forgetfulness it brings! and yet it is but yesterday that one among us taught us its power in arresting certain forms of inflammation. Opium, one of the oldest medicines in use, is still on trial. Clark, even now, superintends its use at Bellevue, and shows the young physician that with it he can reduce the respirations to seven, and even to five, in a minute, and thus hold back the dart of the destroyer till the inherent power of nature comes in to assist in the restoration of the patient to her family and friends. Possibly, we do not even yet know all about opium.

The use of ammonia as a remedy may be as ancient as that of opium, but of that we are not assured, for, according to "Stille's Therapeutics," "The sal ammoniac of the ancients is supposed to have been rock salt, and to have derived its name from the circumstance of its being procured near the temple of Jupiter Ammon of Lybia.

"The temple itself was called after the province Ammonia, in which it was situated, a name which signifies sandy. In the middle ages muriate of ammonia was known as sal armoniacum, or Armenian Salts, in reference to one of its commercial sources. The Arabian physicians speak of its preparation from the soot made by burning (camel's) dung; of its application to the eye for the removal of leucoma; of its use to cure relaxation of the palate, and of its power

of determining the humors to the surface of the body. They also refer to its being mixed in a liniment of oil and vinegar, for the cure of itch. In modern times there is but little recorded of its use as a medicine until the last century, when it became a favorite remedy with German physicians, and continues to be regarded by them as in many cases a profitable substitute for mercury, antimony, or iodine."

In the fall of 1851 my attention was drawn to the use of muriate of ammonia by reading in "Watson's Practice of Medicine" an account of his use of this salt in a certain form of face-ache which he distinguishes from neuralgia and tic douloureux, and then says: "I allude to this for the sake of saying that some years ago I was instructed by an experienced old apothecary that this face-ache might be almost always and speedily cured by the muriate of ammonia; a medicine that is seldom given internally here, although it is so much used in Germany; and I have again and again availed myself of this hint and been much thanked by my patients for the good I did them with this muriate of ammonia." Dr. Watson gave it in half-drachm doses three or four times daily in solution. As my object in this paper is to bring this practical subject before the profession in a strong light, and give all the information I possess of the curative power of this valuable remedy, I do not know that I can do so more readily than by putting my own experience in the form of a narrative.

I had just been appointed visiting physician to the Northern Dispensary, and I had abundant opportunity of testing the muriate of ammonia, not only in the face-ache described by Dr. Watson, but also in other forms of neuralgia, even when of malarial origin. In most cases I was delighted with the speedy relief it

afforded. I was myself a martyr to the form of hemicrania called migrain, and frequently have been obliged to leave my work on account of it, and go home and take one or two doses of half a drachm each at an interval of half an hour, after which I was generally able to resume my duties. I had during that fall a number of typhus-fever patients, and I noticed that many of them, on the second or third day after taking to bed, became unconscious and had low muttering delirium, etc., the usual symptoms of ship fever. It occurred to me that the muriate of ammonia might relieve these symptoms; I used it and I believe with salutary effect; it would frequently arouse them to consciousness. I gave ten grains in solution every half hour with beef tea and brandy, till the patient would awake and be able to answer questions. I believed also that those treated with the ammonia were less liable to inflammatory complications, and that it had a permanent and happy effect till convalescence was established. This experience seemed to me to prove that this agent had a power not generally known, and that it must act on general principles, and I determined to test it in other and different cases. During the following winter there was an epidemic of scarlatina throughout my district, of a mild type, which I treated, as my predecessor had done before me, with chlorate of potash and anointing the body with lard. The success was remarkable, for out of more than 170 cases I reported but three deaths. It seemed to me then that this was nearly a perfect treatment for this usually dreadful disease, but the following year there was another epidemic of more limited extent, but the mortality was frightful. In my despair I sought other remedies, and it occurred to me to add muriate of ammonia to the chlorate of potash, and the result was eminently satis-

factory, for the disease was certainly more under control with this combination than with the chlorate of potash alone, especially when the treatment was commenced early, in the anginous form; the enlargement of the glands and tumefaction of the neck were less, and there was less tendency to deep ulceration in the throat. Its effect in neuralgia about the head, and also its effect in typhus fever, determined me that if sun-stroke or insolation should come again under my care, I would use, with hope for relief, muriate of ammonia. During the summer of 1852 a number of cases of sun-stroke occurred in my practice, and I treated them with this salt, in solution, in ten-grain doses every fifteen minutes. The result was happier than I had dared to anticipate; all the cases treated with the ammonia, when not actually moribund, speedily recovered. Many of my medical friends also used the muriate of ammonia in insolation with happy effect. I furnished a very imperfect account of the cases which I treated during that and the following year, which was published in the *N. Y. Journal of Medicine* for 1854.

Having, in the foregoing experiments, satisfied myself of the power of muriate of ammonia to effect vital changes in the human system when under the influence of disease, I conjectured that it must be by rapid absorption into the blood, and thus by being carried into every part of the body, and by being brought into contact with the capillary nerves, it, in some unexplained way, changed the altered condition of the blood, and at the same time controlled the circulation. In explaining these views to my associates at the Northern Dispensary, I stated that should Asiatic cholera come again into my hands I should expect happy effects from the use of the combination of muriate of ammonia and

chlorate of potash. It was not long before an opportunity was afforded me. On the 23d of May, 1854, I was called to see an Irish emigrant who had landed the evening before, and was then staying with friends living in the rear of 86 Seventh Avenue. He had cold tongue, sunken eyes, sodden fingers, with frequent discharges from the bowels, which his attendants told me were bloody, and they said he had dysentery. I was unable to make a clear diagnosis at the time, but prescribed calomel and opium, and made an appointment to call again next day; but the family becoming frightened took him to hospital, and he died on the way thither. On the 25th of May I was called to the same family to see a little girl ten years old, and found her in collapse. Mustard was applied externally, and stimulants were attempted to be given by the mouth, but she died a couple of hours afterwards. The following day I was called to see the mother of the child, and found her exhibiting the usual signs of cholera. I hesitated to give her the mixture of muriate of ammonia and chlorate of potash, and prescribed, instead, acetate of lead and opium. She died the next day. I now resolved that the next case should have the benefit of the mixture of chlorates. In a few days I was called to see a German emigrant on the corner of Tenth Avenue and Twenty-first Street, and found him in a back basement, badly lighted and without ventilation. He was in collapse, was vomiting frequently, and had rice-water discharges from the bowels. I prescribed the following mixture; R.—Ammon. murias., drams 2; potass. chloras., dram 1; aqua camph., oz. 4; spt. eth. nit.; tr. opii camph. āā., oz. 1. S.—Tablespoonful every half hour.

When I visited him in the evening of the same day the vomiting had ceased, there was sensible reaction,

but he still had occasional passages from the bowels. The next morning he was convalescent. After this I steadily used this mixture in cholera with gratifying success. Some of the gentlemen connected with me at the Northern Dispensary also used it and were pleased with its effects. It evidently stimulated the secretions, especially those of the liver and kidneys, and its effect on the circulation in collapse was notable. The late Dr. Cammann told me that he was called up in the night, that summer, to see one of his neighbors in Fourteenth Street, in consultation with the attending physician. The patient was in collapse and was sinking. Dr. Cammann advised the mixture of muriate of ammonia and chlorate of potash. The pulse was absent below the bend of the elbow, but after taking a dose of the mixture it could be felt creeping again down the artery to the wrist, when after a little while it would again disappear. This fact was noticed by both physicians for an hour or two, but in the end the medicine ceased to have its effect, and the patient died. In many of the successful cases under my care it was the only medicine given, whilst in others it would be instantly rejected from the stomach, and persistence in its use had but little effect till after the exhibition of a full dose of calomel, when the mixture would be retained, and as far as I know there were no bad results from the use of the two remedies at the same time.

About this time I learned from my friend Dr. G. C. E. Weber of the use of muriate of ammonia among German physicians in bronchitis and throat affections, and I began its trial in treating these diseases in combination with chlorate of potash, and was pleased with the result. In croup I had been in the habit of using large doses of calomel according to the method of Dr. Bay

of Albany. In many cases it was speedily successful in arresting the disease, in others a larger amount of mercurial had to be given, and in one case, at least, where, although the croup yielded, consequences followed that caused me to hesitate in repeating the treatment, and subsequently I tried the mixture of ammonia and chlorate of potash instead, and I was surprised as well as delighted to find its power as an antiphlogistic and defibrinating agent quite as manifest as that of calomel, without any of its danger.

In two years I noted twelve cases of croup in dispensary and private practice treated with the mixture with but one fatal result. In all of these there were inflammatory symptoms, and I considered them all to be true croup, although I had the positive evidence of seeing membrane in but two or three instances; still there was a marked difference between these and false croup.

A little girl, five years of age, the daugnter of one of my neighbors, had been suffering with hoarse cough two or three days, and was given domestic remedies, as it was considered only a cold, but at four o'clock in the morning she became so much oppressed with croupy cough and breathing that her father, becoming alarmed, called me up. The cough and breathing were characteristic of croup, the skin was hot and dry, the pulse full and frequent, the fauces were reddened, but there was no appearance of membrane. I sent for the following mixture: R. ammoniæ muriat., drs. 3; potass. chlorat, dr. 1; aqua-cinnamon, oz. 2; syr. g. acaciæ, oz 2; syr. senegæ, oz. 1; and gave her a teaspoonful every five minutes, staying with her until she had taken it a number of times; then, instructing the father to continue it in the same way until there should be either evident relief or vomiting, I went home. At eight

o'clock a.m., I saw her again; the cough was still hoarse, but was accompanied with moist rattles. The father told me he had continued the remedy as ordered for about two hours, when there was coughing with strangling, and he showed me the basin containing the ejected matter; floating in mucus were pieces of ragged, softened membrane, one of them about two and a half inches long, and a little more than half an inch wide, and there was also what appeared to be the detritus of membrane. She had croupy cough throughout the day, and the medicine was given every hour or two, but the next day she was fairly convalescent. I cannot doubt that this was a case of true membranous croup, and as no other medicine was used, the effect of the mixture as a defibrinating agent was, so far, positive evidence. Such happy results in so short a time, however, are the exceptions and not the rule. Usually a longer continuance of the mixture is necessary before the appearance of loosened membrane is manifested.

Sometimes in croup, as in cholera, the mixture had no other effect than to irritate the stomach until after a large dose of calomel was given.

A boy twelve years old, at the Protestant Episcopal Orphans' Home and Asylum, was noticed to be croupy on Wednesday, and was told by the matron to take the mixture, which is always kept in the institution ready for use; he did so and seemed to be relieved. On Thursday evening he was again croupy, and was again ordered to take the mixture; being old enough to wait on himself, he was not watched, and as the medicine was very disgusting to him, he took it sparingly. On Friday morning all the croupy signs were increased. It was the day for the ladies to meet and sew. A messenger was sent to me, but in the meantime, at the suggestion of many of the sympathizing ladies, he was

given Coxe's hive syrup, and syrup of ipecac and squills, alternately, till when I arrived his stomach would keep nothing at all. I immediately gave him a large dose of calomel, after which he took the mixture and retained it. He was in a state of excitement with a constant cough of a ringing, brassy character; breathing was difficult and he spoke only in a hoarse whisper; the fauces were red but no membrane could be seen. The medicine was continued at frequent intervals all night, and on Saturday morning he was spitting up small pieces of softened membrane; the breathing was less difficult and the cough had lost its brassy character, though still somewhat croupy. The medicine was continued through the day and the next night, but at longer intervals, and there were more or less evidences of expectorated membrane, till Sunday morning, when he seemed much better; the croupy cough was gone, he could speak in his natural voice and his breathing was but little affected. The medicine was discontinued and he was ordered nourishing food alone. Still the boy was very much depressed in spirits and expressed his belief that he would never get well. About two o'clock on Monday morning I was called in haste and found him with livid lips and cold extremities, struggling for breath; while flapping rattles were heard over the chest; still his voice was not gone. He died in about half an hour after I arrived. This was a case of true membranous croup. The mixture of muriate of ammonia and chlorate of potash had but little effect till after the exhibition of the calomel, and then its action as a defibrinator was clearly manifested. On Sunday morning the larynx and upper part of the trachea, at least, were cleared of membrane, and the fatal onset of suffocative dyspnœa was owing to occlusion of the smaller bronchiæ, either from membrane

becoming loosened or from the bronchia being closed with tenacious mucus. It was probably an acute case of plastic exudation within the pleura. I could mention many other cases of croup treated with the mixture of muriate of ammonia and chlorate of potash, all showing more or less power of the remedy to relieve the little sufferers, but I deem these two cases sufficient to establish its value, as they are in a good degree a type of the others.

In 1859 diphtheria made its appearance in New York. I had diligently read the British medical journals, noticing the many communciations describing the disease and relating the effects of the different medicinal agents used in combating it; a careful study of these cases had produced in my mind the conviction that the most effectual medicines employed were the chlorates in some form, and especially the chlorates of soda and potash with the muriated tincture of iron. Consequently I was prepared to use what my experience leads me to consider by far the most effectual combination of chlorates, the mixture of muriate of ammonia and chlorate of potash.

I treated the first cases that came under my hands with the mixture, and I was not disappointed in the good results I had hoped from it. I sometimes added to the mixture muriated tincture of iron, and sometimes gave iron and quinine in another form separately, always giving stimulants and nourishment, but the benefit of the mixture was notable, and occasionally marvellously prompt in removing membrane from the fauces in a few hours, but generally about two days of medication was required, while in some long and persistent treatment was necessary. I saw it both in private and dispensary practice, and it appeared as an epidemic several times at the Orphans' Home. I

varied the treatment myself by using that which had been much praised by others, and watched the effect of other modes of treatment in the hands of other practitioners, but I have not yet seen any one form of medication that in my estimation filled all the requisites for success so well as the mixture of ammonia and chlorate of potash.

There is a form of diphtheria in which the tendency is for the membrane to extend into the larynx and air passages, and has been termed, I think properly, diphtheritic croup. When the membrane appears in the air passages below the epiglottis it differs in no way, so far as I know, from the membrane of croup, and I consider it quite consistent with the existing facts that there should be true diphtheritic membrane above the epiglottis and true croup membrane below, during the same attack. No one who has seen much of this disease need be told that when in a case of diphtheria the voice becomes hoarse and whispering, the breathing difficult, and the cough croupy, that the case is one of great gravity, for these signs indicate the presence of membrane in the larynx; in fact they are the signs of membraneous croup.

I have seen cases in diphtheritic croup, as in true croup, get well using no other medicine than the mixture of muriate of ammonia and chlorate of potash, but I have also seen others die under the most persistent use of this medicine.

On account of the disease being diphtheria, I had hesitated to use calomel, as I had done successfully in true croup, but a number of unfortunate cases determined me to use more decided measures; to give calomel and tartarized antimony in combination, in one or two doses, and, after thus forcing an entrance into the system to complete the treatment with the muriate of

ammonia and the chlorate of potash. Such a case occurred to me in January last. A little girl, eleven years old, had sore throat and swollen tonsils on the 18th of January. She was given the mixture. On the 20th of January membrane covered the tonsils, and was continuous over the walls of the pharynx. The mixture was ordered in larger doses, and at more frequent intervals. The pulse was full and bounding, for the child was naturally robust. On the evening of the same day the symptoms had rapidly grown alarming, the voice was husky, and the breathing was becoming difficult. It seemed to me that the ammonia and chlorate of potash did not enter the circulation. I prescribed two powders, each containing two grains of calomel and one-sixth of a grain of tartarized antimony, with ten grains of pulverized sugar, to be given at an interval of three hours, the mixture to be given in the meantime every half hour, one tablespoonful. In the morning she was weary, but the voice was clear, the breathing was improved, and the appearance of the fauces was changed, being of a brighter red color, and the membrane was becoming detached. She continued the mixture one tablespoonful every two hours, and made a rapid recovery, for on the 22d she was fairly convalescent.

The following notes were made by Dr. Cummings, the able House Surgeon of the Demilt Dispensary, in two cases lately occurring in his practice, and as they are independent testimony, coming from an observer without theory or prejudice, I offer them as corroborative of the value of the mixture of muriate of ammonia and chlorate of potash as a remedy in serious forms of diphtheria.

*Case* I.—*Diphtheria affecting the larynx terminating in recovery.*—December 25th, 1863. Saw for the first

time a boy, August Weber, aged three years and four months, who had been ill for four days, complaining of symptoms referable to the throat. It was eleven o'clock at night when I first saw him; parents stated that he was much worse this evening than he had been previously. Croupal respiration and cough were both well marked; face expressive of much anxiety and lips livid; pulse 120 per minute and weak. The submaxillary region was much swollen; the voice also hoarse and indistinct. On opening the mouth the tonsils were seen to be tumefied and covered by a false membrane of a whitish color; the pillars of the palate were likewise covered with false membrane.

From the fact that the disease was so advanced and the laryngeal symptoms so severe, an unfavorable prognosis was given. The child was ordered four grains of the chloride of ammonium and one and one fourth grain of the chlorate of potassa every half hour, in a teaspoonful of camphor water, also five drops of the chloride of iron every four hours. Fomentations were likewise directed to be applied to the neck.

December 26th, 9 A.M. Found the patient a little more comfortable, but the fauces presented pretty nearly the same appearance as on the previous night; the face was very pale, but had not quite that lividity which was observed at the former visit; child took liquid food greedily, and had experienced great desire for sleep during the night. The same medicines were continued, and beef tea and milk punch also ordered. Saw the child again that night; cough and breathing distinctly laryngeal, yet the obstruction to respiration did not seem quite so great as on the preceding night.

December 27th. Patient was decidedly easier; had passed a tolerably comfortable night. Respiration less stridulous; cough had a little more of a moist character;

membranes seemed to have diminished in extent, and to appear thinner and somewhat detached at their edges; appetite still good; directed to continue the same medicine.

On the 29th of December the tonsils and throat had become completely free of the false membranes, and the child was still improving, although the croupal cough remained.

Chloride of ammonium and chlorate of potassa were ordered in the previous doses every two hours. Quinine was also given as the appetite of the child was failing; iron continued.

January 3d. Bronchitic râles were now heard. These disappeared in a few days under the influence of general counter-irritation and expectorants. The croupal cough continued until Jan. 7th, when it had entirely disappeared and the child was dismissed from my care, with directions to take the iron a week longer. Since then I have heard from the child, who remains in perfect health. I would add there were in the house where this boy lived four other children suffering with pharyngeal diphtheria, under my care, at nearly the same time, all of whom recovered, the same treatment having been pursued.

*Case* II.—*Diphtheria involving the larnyx terminating fatally.*—December 30th, 1863. Was called to see Margt. Quinn, aged four years and eight months. This child had been suffering with sore throat five days; could not learn that she had experienced any fever.

This patient exhibited decided stridulous breathing, inspiration and expiration being both very much prolonged, a ringing croupal cough, and the voice was quite extinguished. The lips were livid, the eyes prominent, the head thrown back, and the whole ex-

pression one of great distress. The pulse was frequent and feeble. On inspecting the fauces, a dense grayish white membrane was seen covering the tonsils and pillars of the palate, not patchy, but continuous; there were also bridles across the posterior pharyngeal walls, and the uvula was enveloped by a layer of membrane.

An unfavorable prognosis was made in this case. Death seemed imminent from the obstruction in the larynx.

The patient was given five grains of chloride of ammonium and one grain and a fourth of chlorate of potassa every half hour, in a teaspoonful of syrup and water. Five drops of the chloride of iron were given, in the same vehicle, every four hours; milk punch and beef tea were also ordered. The next day, when the child was visited, its general appearance had a little improved, although it had experienced several attacks, threatening suffocation, during the night. The respiration seemed a little less difficult than on the preceding day; not much change was observed in the condition of the throat. The respiration now continued steadily to improve, and on the 2d of January the membrane was evidently disappearing on all parts accessible to the eye. The chloride of ammonium and chlorate of potassa were now given in half of their previous doses. The iron was continued as before. As soon as the difficulty of respiration was somewhat relieved, the child exhibited a great tendency to sleep, both day and night, showing the severe toxæmic effect of the diphtheritic virus.

On January 4th no membrane was visible, and the breathing of the child had become perfectly calm; all cough had likewise disappeared. There was now noticed on the left tonsil a small perforating ulcer, looking as though it were bored or punched into the gland,

There was also paralysis of the muscles of the palate, occasioning much difficulty in swallowing, producing a cough and regurgitation of food through the nostrils; a muco-purulent discharge, at times streaked with blood, also issued from the nostrils. Quinine, in addition to the iron, milk punch and beef tea, was now given; the chloride of ammonium and chlorate of potassa were discontinued. Jan. 6th, the ulcer continued to increase in extent and depth, and other ulcers were seen starting around the original one; discharge from the nostrils more streaked with blood; moist bronchitic râles were now heard for the first time, apparently not much embarrassing the respiration. Patient continued weak, but took medicine and nourishment very well. For the bronchitis gentle counter-irritation to the chest and stimulating expectorants were employed.

January 8th.* Râles distinctly heard, seemed to involve the smaller bronchial tubes on one side; no dullness on percussion; no great difficulty in respiration; child pale and weak; pulse frequent and feeble; same treatment continued, with injunctions to give an additional amount of stimulants.

January 9th. Visited the child at 12 M., who seemed rather more comfortable than the day before. The child continued quite comfortable, as I understood by the parent, until 6 P.M., when immediately after taking food it died, dropping off as though in a state of syncope.

These two cases seem to me to illustrate the efficacy of chloride of ammonium in promoting the separation

* These râles were undoubtedly inter-pleural plastic, but at that time I had not yet learned their true signification nor had Dr. Cummings

of the diphtheritic membranes as well as in relieving the swollen condition of the parts on which they rest. In the last case the relief to the laryngeal obstruction commenced almost immediately upon its administration, although the child subsequently died of blood-poisoning.

I am in the habit of employing it in all cases of diphtheria, as I know of nothing that answers the above-mentioned indications equally well.

ISAAC CUMMINGS, M. D.

DEMILT DISPENSARY, Feb. 1st, 1864.

I have been constantly in the habit of giving muriate of ammonia, alone or in combination, in all forms of inflammation, not depriving myself, however, of the choice of more actively efficient agents when the cases seemed to require them.

In pneumonia it acts promptly and efficiently, and also in sub-acute pleuritis; in congestion of the brain it frequently affords prompt relief. Even in acute meningitis of children it acts with apparent benefit, lowering the pulse and preventing convulsions. In tubercular diseases of all forms I deem it decidedly beneficial, and especially in phthisis. During the last five years I have had large experience with the muriate of ammonia as a remedy in tubercular phthisis at the Demilt Dispensary, in the class of chest diseases, with the result of confirming my confidence in its remedial power. No other single agent has been so beneficial in my hands. I prescribe it with wild cherry bark in cold infusion given at frequent intervals.*

I believe muriate of ammonia to be essentially a blood medicine; it must enter the circulation to pro-

* ℞ Ammon chlor. ʒ i. cortex. P. Virgin, ʒ ij. M. Cold infusion by percolation two pints, S. one tablespoonful every hour.

duce its effect, and this is the only explanation I have to offer for its apparent benefit in diseases of such opposite types. I believe it acts as a catalytic and also as a resolvent; that as a catalytic it accomplishes its work of arresting inflammatory action without any such destruction of blood corpuscles as is done by mercury.

Mialhi estimates that one-third of the blood corpuscles of the body are destroyed by placing the system under the influence of mercury. If that be true, chloride of ammonium is much the safer agent, especially in debilitated constitutions. As a resolvent it is believed by German physicians to act upon glandular swellings and recent tubercle, and my favorable experience with it leads me to adopt that view.

I have mostly used it as an internal medicine, but in some cases I have thought it produced good effects in the bath. "Dr. Giesler used it in the form of vapor by inhalation in chronic catarrh, and never found it useless." He also recommends it in some forms of rheumatism, and in strumous ophthalmia. Dr. Noeggerath, of New York, has used the vapor of muriate of ammonia successfully in some cases of diphtheria. It is readily vaporized by placing it on a hot metallic surface, and it strikes me that this mode of using it, in some cases at least, must be preferable to any other.

Some years ago, Dr. Batchelder, of New York, mentioned to me that the iodide of potassium was more energetic and produced its characteristic effects in much less time than usual, when mixed with an equal or larger amount of chloride of ammonium. I have satisfied myself many times since of this fact, and also that it energizes the action of other remedies when in combination, as in chlorate of potass., nitrate of potash and the muriated tincture of iron. A mixture of muriate of ammonia, nitrate of potash and senega root,

colored with cochineal, is sold as a common remedy for influenza or cold in the head, I am told, from the drug-shops in the towns along the upper part of the Hudson River. It was a favorite prescription of the late Dr. White of Hudson, and is known as "White's Red Salts." Half an ounce each of these articles, with liquorice root to disguise the taste, may be infused in a pint of water; dose one tablespoonful every fifteen minutes for an hour or two before going to bed generally relieves a patient with commencing influenza, and he awakes in the morning well. All surgeons are aware with what energy a saturated solution of muriate of ammonia and bichloride of mercury will act as an escharotic.

Muriate of ammonia has been held in high estimation by German physicians for more than a hundred years. At the close of the last century Gmelin said of it, that "it is by far the most powerful of saline preparations, whether as an internal or external agent." "Bocker considers its therapeutical action to depend upon its quickening the moulting or waste of mucous membrane, and on this account its protracted use in young people especially is to be avoided." This view I believe to be mere hypothesis, not borne out by experience. "Osterlin states that by mistake one of his patients took two ounces of muriate of ammonia at a single dose without any other result than trifling colic and some watery stools." "It is praised by Gmelin for its efficacy in intermittent fevers." "In 1851 M. Aran experimented with it and considers that the results indicated that it possessed some and not a little power over intermittent fevers." "Jacquot, also, in 1851–2, used it in treating soldiers of the French army occupying Rome. The results consisted in the abrupt cessation of the paroxysms in six out of twenty-

one cases, but in two of the six cases the attacks returned."

In 1855 Dr. Alexander Lindsay published in the Glasgow *Medical Journal*, an article on the "Physiological and Therapeutical effects of the Chloride of Ammonium." "Dr. Lindsay and two intelligent pupils made experiments on themselves, taking the chloride in medicinal doses, being in a state of health, and carefully regulating their diet, etc. On the second day after beginning the medicine a buoyancy of the system was experienced that rendered the ordinary pursuits a pleasure, and fitted the body and mind for increased exertion." "The feculent discharges were in all much augmented, the appetite was much improved. In two the force and frequency of the heart's action were diminished. The rate of the pulse in the gentleman employing the smallest dose was accelerated. In all the urinary secretion was increased. The dose was, in one 18 grains per day; the second, 13½ grains, and the third nine grains." This is the only record that I am aware of in which experiments have been made with chloride of ammonium on healthy persons. Dr. Lindsay used the remedy in many and various diseases, and is much pleased with the results. He combined it with tartarized antimony and morphia. Dr. Walshe says, "Muriate of ammonia has appeared to me to be useful in two apparently opposite ways—by promoting expectoration when deficient, by controlling its amount when excessive." In the "Astley Cooper Prize Essay," for 1856, on "The Cause of Coagulation of the Blood," by B. W. Richardson, M.D., it is shown by a number of experiments that fresh-drawn blood gives off free ammonia during the process of coagulation. It is also shown that the addition of ammonia to the blood retards the coagulation according to the amount

used; that ammonia added to coagulated blood will cause it to again become fluid, and that it will again become coagulated when the added ammonia has passed off in vapor. "That ammonia is evolved from the blood," says his reviewer, "on its being withdrawn from the vessels and exposed to the air, has been proved most satisfactorily by Dr. Richardson's experiments, which have been so multiplied and varied as to exclude all sources of fallacy."

These experiments go to show that ammonia is necessary to healthy blood; that in excess it is rapidly thrown off in the excretions, and in this way it is not allowed to accumulate unduly; that ammonia, taken into the system in whatever form, is thrown off as free ammonia, and this may explain why its combination with other agents so increases and energizes their characteristic effects.

Dr. Ozier Ward, in the London *Lancet* for April 1859, says: "Ammonia had never been considered to be a normal constituent of the blood, as its presence had not been detected except after death, in cases of typhus, cholera, melæna, and other diseases of a putrid character, until Dr. Richardson's recent discovery that healthy blood owes its fluidity to the presence of ammonia." In speaking of its therapeutical effects, he says, finally: "The hydrochlorate, which is the least easily decomposed, is probably the most useful of the salts of ammonia, as it not only possesses the stimulant, resolvent, secernent properties of the others, but, owing to its combination with chlorine, is endued with tonic powers, by which its prolonged use, unlike that of the other preparations, is attended with invigorating effects both to mind and body, and that it forms an excellent substitute for mercury, in cases where this

medicine is inadmissible from its tendency to produce cachexia."

Perhaps this record of my own experience, with notes of that of other observers at different times and in different places, may help to show that muriate of ammonia, known to the ancients, much valued by the Arabian physicians of the middle ages, and again introduced into practice by German physicians a century ago, is still upon trial, and that facts are accumulating which promise to elevate it into a prominent place in our pharmacopœia.*

* After so many years since the publication of this article I have it still in constant use. In cold infusion of wild cherry bark, sixteen to twenty grains to the ounce, half ounce doses of the mixture, it is of great service in inter-pleural plastic exudation, and in the early stages of fibroid phthisis. Many cases get well with no other medication. When this article was written, muriate of ammonia was used in medicine in New York only in washes and liniments. It is in such general use now, and so favorably, that its advocacy in this article seems strange and unnecessary.

## ARTICLE II.

### Therapeusis of Mercury.

The physician needs powerful medicines to control disease; none the less because he believes in "vis medicatrix naturæ." We require of the surgeon that his knives be sharp and that he have skill to use them—that he use them not on wrong or slight occasions. In the armamentarium of the physician there is no other agent having the powerfully sedative and at the same time the delicately alterative effects which belong to the different preparations and doses of mercury.

It has been said of the steam-engine that its adaptativeness is universal. It can be made to engrave the delicate tracery of a seal, or to lift a man-of-war out of the water. We may say the same of electricity—its power is unlimited, its power and adaptativeness to nice results is marvellous. So also may we say of mercury as a medical agent for the control of diseases. Yet there is no other remedy against which there is such a violently unreasoning and unwise prejudice as against mercury, especially against the most useful of all its preparations—the mild chloride, calomel.

How absurd would be popular prejudice against the steam-engine or against electricity? Are they not powerful for destruction of human life if misdirected? Yet they are our obedient servants for good under intelligent direction. So is calomel.

Calomel may be given in drachm doses, and save life when no other remedy can do it, and no harmful result follow. It may be given in one-hundredth part of a

grain doses with the nicest ascertainable effects. It simply needs to be wisely adapted to the necessities of the case for its exhibition.

Pleuro-pneumonia, which has prevailed for twelve or fifteen years epidemically in New York, is controllable in the worst cases only by the sedative action of calomel.

This agent is the shears that may clip the locks of the destructive Samson.

Dr. Graves, on large doses of calomel in acute inflammation, says ("A System of Clinical Medicine," Dublin, 1843): "The following remarks derived from very extensive opportunities of observation apply not to the treatment of chronic diseases, nor to that of inflammations, either slight in degree or occupying parts not essential to life, but to those violent attacks of inflammatory action which so often prove fatal in the course of a few days or even hours by destroying the texture and function of vital organs.

"If a person is seized, for example, with very acute pericarditis, how unavailing will be our best directed efforts unless they be seconded by a speedy mercurialization of the system. If, on the contrary, the practitioner defers the exhibition of calomel *or insufficiently uses it*, then will he have occasion to regret the consequences, and witness either the speedy death of his patient or his condemnation to the sufferings entailed on him by adhesions, valvular disease, and other sequelæ of badly-treated pericarditis."

I well remember my astonishment when thirty years ago the late Dr. G. P. Cammann ordered a large dose of calomel in an attack of intercurrent pneumonia in a case of chronic phthisis; and my gratification at seeing the disease successfully controlled thereby. It was, perhaps, the most practical of all the valuable lessons which I received from him.

Dr. Graves considered the speedy mercurialization of the patient as necessary He quotes Dr. Johnson's classical work on the "Diseases of Tropical Climates," which says "we ought to affect the constitution decidedly and as speedily as possible by means of calomel, given, not in small doses often repeated, but in doses of a scruple, once or even twice daily."

But in the *sedative* action we do not contemplate *mercurialization* in the sense of ptyalism or salivation. And if that should occur it is accidental and is due to an unfortunate idiosyncrasy of the patient.

The admirably sedative effect of calomel when needed is best seen when it is placed dry upon the tongue of the patient; then, like the touch of the wand of the magician, it instantly changes the conditions of death to those of life. There is no absorption of the medicine, no exhausting purgation, no salivation.

The temperature at once begins to fall, the heart to gain strength, the plastic exudations upon vital organs to be reabsorbed, and the course of life again runs smoothly on. Of course it should not be given in any case where simple means would answer.

We may say the same of any medicine. But some forms of inflammation of vital organs; of the brain, of the heart, of the lungs or kidneys, or some forms of dysentery or fevers, may be speedily fatal, if not arrested early in the attack. In that supreme moment there is no choice; there is but one remedy. If the physician hesitates then, or searches for other remedies in obedience to popular prejudice, the favorable moment may pass and the patient be lost. But surely the accident of salivation is nothing, even when severe, in comparison with the death of the patient. Loss of teeth, or necrosis of the jaw, or cancrum oris, do not follow the use of the *sedative action of calomel.* Such

untoward results come from the poisonous effect of calomel, given in repeated smaller doses. There was a time when abuse of this powerful remedy was not uncommon. But such is not the case now. The accident of salivation, which may occur when one or two large doses may be necessary, is not destructive to tisues, bones, or teeth. It is simply an annoying inconvenience.

The poisonous effect of mercury is not its sedative effect. Any one who has seen twenty, thirty, or even sixty grains of calomel placed on the tongue, at the right time, in a case requiring its use, cannot help being gratified at its beneficence and its power to save. It has no unpleasant effect, simply the patient gets well; and the change is so quiet and so complete that we feel doubt almost that there ever had been such danger.

When in the judgment of the physician the time has arrived for the use of this great remedy, it should not be delayed, and the dose should not be scrimped. The dose should be ample. Our fears of public prejudice make us cowardly, and we sometimes make the mistake of giving too little, and so may do harm. The small dose at that time is dangerous in letting the time for successful action pass by. It may have to be repeated, and the poisonous effect of mercury may take place. There is no danger in the largest dose when it is needed.

Small doses given in combination with opium, may be very serviceable. Calomel one half a grain, with five grains of Dover's powder, may be of decided benefit, given according to the needs of the case in progressive inter-pleural fibrination, or fibroid phthisis.

But the combination of calomel, tartar emetic, and nitrate of potash, mentioned by Dr. Rush in 1800, as the fever powder of Pa. Genl. Hosp., and which he

used in treating successfully what he called consumption in the third stage, is admirable in fibroid phthisis of any stage.

This combination may be given with effect when the calomel may not exceed the one-hundredth of a grain. In the Polyclinic Dispensary we have this combination ready in the form of tablets for convenience.

The stronger tablets (No. 3) contain one-fifth of a grain of calomel, one-thirtieth of a grain of tartar emetic, and five grains of nitrate of potash. The tablet is made up with sugar, gum acacia, and licorice.

The second in strength (No. 2) is just half the amount of the first, and the third (No. 1) one-fourth. They are allowed to dissolve on the tongue.

Bichloride of mercury dissolved with muriate of ammonia, in Huxham's tincture of bark, is also a very serviceable combination, and may be given alternately with iodide of potash, as in syphilis. Fibroid phthisis is frequently the result of syphilis. But whether a given case is so or not the treatment is equally beneficent.

Mercurial inunction I have used more formerly than at present. It is not so manageable and the dose is not so sure as when given by the mouth or on the tongue. But it can be used, as may also the mercurial vapor, in some cases with singular benefit.

## ARTICLE III.

### Thuja Occidentalis.

Arbor-vitæ, or American white cedar, has for more than a hundred years been a remedy in use for a variety of ailments. It grows indigenous over the Canadas and the United States. The terminal twigs and green leaves may be made into a tincture with alcohol (95 per cent).

From this a fluid extract or an elixir may be formed, and used as a medicine. As an ointment or as tincture it has been applied to indolent ulcers, to warts, and to polypi with supposed benefit. The tr. or fluid ext. applied to an indolently inflamed pharynx, with engorged tonsils, on cotton or by the spray, gives immediate relief. A method of applying it is to wind some cotton batting upon the end of a wire or a probe, and charge it with the tr. or fluid ext., then requesting the patient to take a full breath, and while holding the mouth open, to quickly pass the charged cotton over the tonsils and pharynx. Upon withdrawing the probe let the patient shut his mouth and breathe slowly out through the nose.

When there is laryngeal and nasal catarrh combined with engorgement of the pharynx the vapor reaches distant parts in the nasal passages in breathing out, as well as in the larnyx in breathing in, and gives relief. The engorgement and color of the pharynx and tonsils are instantly affected.

This remedy has been used with supposed benefit in certain forms of malignant diseases characterized by

engorgement and hemorrhage. I have seen cauliflower excrescence disappear in a short time under its influence, and it seems to arrest the tendency to bleed. In the early stage of fibroid phthisis characterized by sudden attacks of congestion, hæmoptysis, and plastic exudations within the pleural cavities, I have seen these alarming conditions disappear in a very short time while giving the patient twenty or thirty drops of the strong tr. or the fluid ext. on sugar or in oil or in cream every three or four hours. When the pulmonary congestion is complicated with suppression of the menses the exhibition of thuja may give relief to both conditions speedily.

I have known cases of pulmonary engorgement, with hæmoptysis, with moist and abundant râles over the chest, to be greatly relieved with two or three days' use of the thuja supplemented with terebinthinate applications externally. The abundant moist râles disappearing so speedily would seem to indicate that this remedy has power over recent plastic exudations for their removal, and in this way arrest hemorrhage. Although not a specific for cancer, or tubercle, or fibroid, so far as I know, it may be found to be of great service in controlling these diseases by relieving the system of hyperæmia and the hemorrhagic tendencies.

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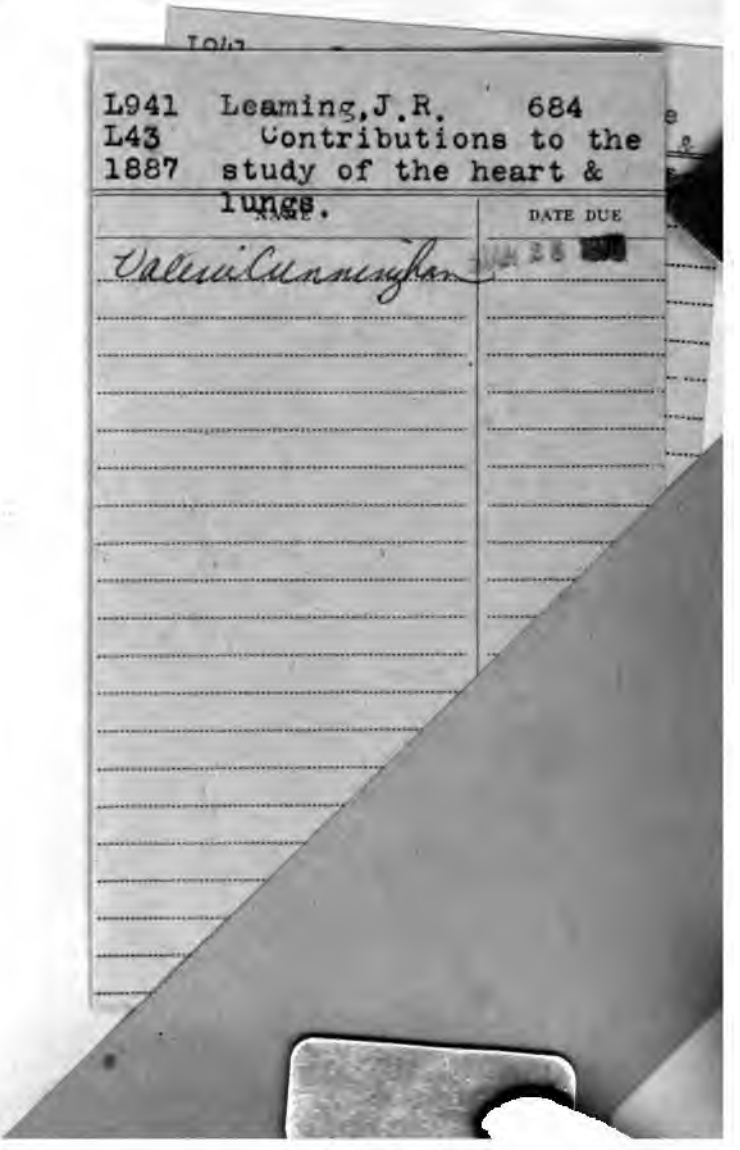
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